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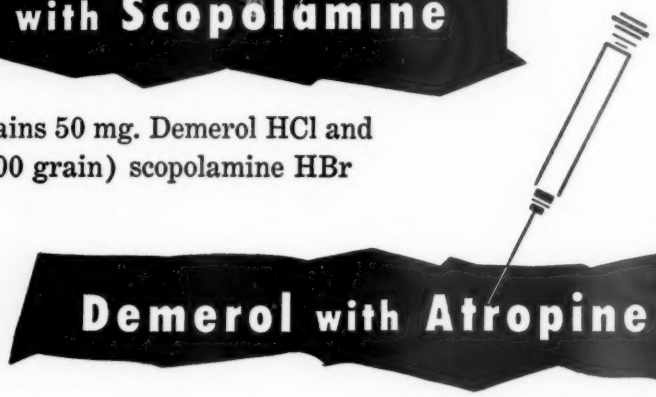

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SURGERY

VOL. 41 MAY, 1957 NO. 5

Original Communications

DIAGNOSIS OF LUNG CANCER BY BRONCHOSCOPIC BIOPSY, SCALENE LYMPH NODE BIOPSY, AND CYTOLOGIC SMEARS

A REPORT OF 42 HISTOLOGICALLY PROVED CASES

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INTRODUCTION

THE importance of bronchoscopic biopsy and, more recently, of anterior scalene lymph node biopsy in the diagnosis of lung cancer has been well documented. The examination of sputum and bronchial secretions for the presence of malignant cells has also been found to be of practical value by most investigators and the method is now almost universally accepted.

The purpose of this study was to determine the relative sensitivity of each of these diagnostic procedures and to investigate any relationship between these methods and the location, histologic type, and resectability of bronchogenic carcinoma.

METHOD AND MATERIAL

During the period between January, 1954, and January, 1956, 42 patients treated at the Veterans Administration Hospital, Ann Arbor, for histologically proved bronchogenic carcinoma had diagnostic cytologic studies of their sputum, bronchial secretions, or both. Patients with clinical evidence of lung cancer which was not confirmed histologically were excluded from this study. Thirty-eight of the 42 patients had bronchoscopic examinations and 29 had anterior scalene lymph node biopsies. The latter were performed according to the technique described by Daniels[4] and were obtained from the right side for all lesions in the right lung and the left lower lobe, left scalene node biopsies being reserved for lesions in the left upper lobe and its lingular segments.

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The cytologic smears were prepared from sputum or bronchial secretions in accordance with the Papanicolaou technique.[10] Four smears were prepared from each specimen. A specimen was not reported as negative until a minimum of 5 to 20 minutes (depending upon the cellularity of the preparation) had been allotted to its microscopic examination, although positive smears frequently required less time for examination. The results were classified as positive, suspicious, or negative, and the microscopic type of cancer was stated in most of the positive and suspicious reports. The findings have been summarized in Tables I to IV.

RESULTS

Bronchoscopic Biopsy.—A bronchoscopic examination was interpreted as positive only when bronchial tissue was obtained and the histologic examination of the latter permitted a microscopic diagnosis of carcinoma. Bronchoscopic examinations were performed in 38 of the 42 patients. Carcinoma was demonstrated in 15, or 40 per cent, of the 38 patients (Table I). Thirteen positive biopsies were obtained from 23 patients who had squamous-celled carcinoma and 2 from 7 patients with undifferentiated-celled carcinoma. None of 8 adenocarcinomas were diagnosed by this method.

TABLE I

		BRONCHIAL BIOPSY		SCALENE NODES		CYTOLOGIC SMEARS		
HISTOLOGY	NO.	POS.	NEG.	POS.	NEG.	POS.	SUSP.	NEG.
Squamous-celled	25	13	10	1	15	18	5	2
Gland-celled	8	0	8	3	3	5	1	2
Undifferentiated, small-celled	5	1	2	2	2	3	1	1
Undifferentiated, large-celled	4	1	3	1	2	3	1	0
	42	15	23	7	22	29	8	5
False negatives			60%		76%			12%

Positive bronchoscopic biopsy was found in 5 of 7 bronchogenic carcinomas which were situated in main-stem bronchi and in 8 of 11 neoplasms involving the lower lobe of either lung (Table II), but only 2 of 19 tumors of upper lobes yielded positive bronchial biopsies. The negative group included a carcinoma of the right middle lobe and another which was at the pulmonary hilum but its lobar location was not determined.

TABLE II

		BRONCHIAL BIOPSY		SCALENE NODES		CYTOLOGIC SMEARS			COMBINED METHODS
LOCATION	NO.	POS.	NEG.	POS.	NEG.	POS.	SUSP.	NEG.	POS.
Main-stem bronchus	7	5	2	2	2	5	2	0	7 (100%)
Upper lobe	22	2	17	4	14	13	5	4	18 (82%)
Lower lobe	11	8	3	1	5	9	1	1	11 (100%)
Right middle lobe	1	0	1	0	0	1	0	0	1
"Hilar"	1	0	0	0	1	1	0	0	1

In Table III the neoplasms have been separated into those which were located in the hilar region and those which were peripherally located. This

distribution was determined by gross pathologic or roentgenographic examination. Bronchial biopsy detected 15 of 24 hilar cancers, but none of 14 peripheral neoplasms, in those patients who had bronchoscopic examinations.

TABLE III

LOCATION	NO.	BRONCHIAL BIOPSY		SCALENE NODES		CYTOLOGIC SMEARS		
		POS.	NEG.	POS.	NEG.	POS.	SUSP.	NEG.
Hilar	27	15	9	3	15	20	5	2
Peripheral	15	0	14	4	7	9	3	3

TABLE IV

	RESECTED	NOT RESECTED
Bronchoscopic Biopsy		
Positive	4 (26.7%)	11 (73.4%)
Negative	12 (52.2%)	11 (47.8%)
Cytologic Smears		
Positive	8 (27.6%)	21 (72.4%)
Suspicious	4 (50.0%)	4 (50.0%)
Negative	4 (80.0%)	1 (20.0%)

Lobectomy or pneumonectomy was performed in the case of only 4 of the 15 patients with positive bronchoscopic biopsies, while the tumors of 12 of the 23 patients who had negative bronchial biopsies were resected (Table IV).

Anterior Scalene Lymph Node Biopsy.—Twenty-nine anterior scalene lymph node biopsies were performed. Seven, or 24 per cent, contained metastatic neoplasm (Table I). The number of lymph nodes found in specimens which contained metastatic neoplasm varied from 1 to 8, with an average of 5. The negative specimens revealed from 0 to 19 nodes per specimen, with an average of 6.

The anterior scalene lymph nodes from 16 patients who had squamous-celled bronchogenic carcinoma revealed neoplasm in only 1 patient. On the other hand, 3 of 6 patients with adenocarcinoma and 2 of 4 patients with undifferentiated-celled carcinoma had positive scalene node biopsies.

Four of the 7 positive scalene node biopsies were associated with carcinomas of upper lobes, 2 with carcinomas of main-stem bronchi, and only 1 with a lower lobe carcinoma (Table II).

Positive scalene node biopsies were obtained from 3 patients who had hilar neoplasms and 4 patients who had peripheral neoplasms, but 15 patients with carcinomas of the hilar portion of the lung were found to have negative nodes, while only 7 patients with peripherally located tumors had nodes which did not contain tumor (Table III).

Bronchoscopic biopsy was positive in only 1 of 7 patients who had positive anterior scalene lymph node biopsy but each of the patients whose scalene nodes were positive had malignant cells in cytologic smears of sputum or bronchial secretions.

The presence of metastatic carcinoma in the anterior scalene lymph nodes constituted the sole criterion of inoperability in 4 of the 7 patients who had

positive anterior scalene node biopsy. The inoperability of the other 3 was suggested by bony metastasis, superior venal caval syndrome, and diaphragmatic paralysis. Incidentally, the scalene node biopsy provided the only histologic evidence of carcinoma in these three patients. The lymph nodes were palpable in 2 of these 3 cases.

Cytologic Smear Diagnosis.—Twenty-nine, or 69 per cent, of the 42 patients had unequivocally positive cytologic smear reports, and the smears from another 8 patients contained suspicious cells which were of insufficient atypism to warrant a definite positive diagnosis (Table I). Eleven patients, 26 per cent of the entire group, had positive smears as the only microscopic evidence of carcinoma prior to thoracotomy.

Twenty-three of the 25 patients, or 92 per cent, with squamous-celled carcinoma were found to have malignant or suspicious cells in smears from their sputum or bronchial secretions (Table I). Cytologic smears from 5 of 8 patients with adenocarcinoma of the lung contained malignant cells and smears from another patient with gland-celled carcinoma were interpreted as suspicious. Only 1 of 9 patients who had undifferentiated-celled carcinoma had negative cytologic smears.

Each of the 7 patients who had a cancer arising in a main-stem bronchus had a positive or suspicious cytologic smear (Table II). Only 1 of the 11 patients who had a carcinoma of a lower lobe had negative smears, but there were 4 cytologic failures in the group of 22 upper lobe tumors. Cytologic smears from the patient who had a right middle lobe carcinoma were positive.

Twenty-five of the 37 positive or suspicious cytologic smears were from patients with hilar carcinomas (Table III). Only 2 hilar and 3 peripheral carcinomas failed to disclose malignant cells in sputum or bronchial secretions.

Eight of the 29 patients who had positive cytologic smears were treated by means of lobectomy or pneumonectomy, while the remaining 21 were not considered to be candidates for resection either because of the extent of the neoplastic involvement or because of other contraindications. On the other hand, the tumors of 4 of the 5 patients with negative smears and of 4 of the 8 patients with suspicious smears were resected.

False Positive Cytologic Reports.—There were 2 false positive cytologic reports during the period covered by this report. This represented 2 errors from a total of 175 patients who were believed to have pulmonary diseases other than bronchogenic carcinoma, a false positive error of 1.1 per cent. Pathologic study of the resected lung from one of these two patients disclosed severe chronic pneumonia and pulmonary infarction with severe atypical squamous metaplasia. The other patient had 1 positive specimen and 10 subsequent negative specimens. It was suspected that a technical error, such as mislabeling of the specimen, had occurred; but it could not be traced. This patient was ultimately discharged with a diagnosis of chronic pneumonia and without having had a thoracotomy.

DISCUSSION

It has been reported that a positive diagnosis of bronchogenic carcinoma may be established in from 26 to 60 per cent of patients by means of bronchoscopic biopsy.[1, 13, 15] The average sensitivity of this technique appears to be between 35 and 45 per cent. In our series, bronchoscopic biopsy was positive in 40 per cent of the patients with primary lung cancer.

Most of the positive bronchoscopic biopsies were obtained from patients with squamous-celled carcinomas and this was attributed to the frequent involvement of major bronchi by this type of lung cancer while the peripheral location of most adenocarcinomas accounts for their frequent lack of detection by bronchial biopsy. It is obvious that this modality is of little value in the diagnosis of peripherally located carcinomas.

The majority of carcinomas of lower lobes or those arising in main-stem bronchi were detected by bronchial biopsy, but the percentage of success decreased sharply in the diagnosis of upper lobe cancers; only 2 of 19 upper lobe carcinomas have been confirmed by this method. This is especially significant since upper lobe carcinomas are as common or more common than those arising in lower lobes.

In 1949 Daniels[4] demonstrated the merits of anterior scalene lymph node biopsy in the diagnosis of intrathoracic disease. Cuykendall[3] found metastatic neoplasm in these lymph nodes in 3 of 14 patients (21.4 per cent) with bronchogenic carcinoma. Using a modification of Daniel's method, Harken and associates[7] increased the percentage of positive results to 39.8 per cent, while Shefts and co-workers[12] reported the successful detection of metastatic neoplasm in 54.2 per cent of their patients who had bronchogenic carcinoma. The possibility of crossed metastases and the importance of bilateral anterior scalene node biopsy in patients with left lower and left midline lesions has been discussed by Harken and his associates[7] and by Connar.[2]

Anterior scalene lymph node biopsy has a double purpose: the histologic confirmation of the presence of a malignant tumor, and the determination of inoperability. Although only 7 patients in this series had positive scalene node biopsies, the latter was the only histologic confirmation of positive cytologic findings in these 7 cases. In addition, the presence of metastatic carcinoma in these nodes constituted the sole criterion of inoperability in 4 of these 7 cases.

Previous reports have indicated that the incidence of metastases to anterior scalene lymph nodes is determined to a large extent by the location and the histologic nature of the carcinoma.[2, 3, 7, 12] Connar[2] obtained positive biopsies in 8 of 18 patients who had upper lobe cancers and negative biopsies in each of 8 patients who had lower lobe tumors. Our findings were similar, all but one of the positive biopsies having been found in association with neoplasms of upper lobe or main-stem bronchi. It was also found that the peripherally located carcinomas metastasized to these nodes in a higher percentage of instances than had those in the pulmonary hilum. The probable explanation

for this observation will be discussed later. It is significant to note that scalene node biopsy is most successful in the lung cancers which are most difficult to diagnose by means of bronchoscopic biopsy, that is, upper lobe carcinomas or carcinomas arising in small or peripheral bronchi. Conversely, the lower lobe tumors which seldom metastasize to the anterior scalene nodes are usually accessible to bronchoscopic biopsy.

Shefts and associates[12] and Connar[2] have noted that bronchogenic carcinomas of undifferentiated-celled type are most likely to give positive scalene node biopsies. Metastases from bronchogenic adenocarcinomas have also been found frequently.[12] In contrast, Shefts and co-workers did not encounter any metastatic squamous-celled carcinomas among their positive cases and Connar reported only 2 specimens which contained this type of carcinoma despite the fact that it was the most common histologic type in his series. These findings have been confirmed by our studies. Only 1 of the 16 patients with squamous-celled carcinoma had a positive biopsy, while 50 per cent of the patients who had undifferentiated-celled or gland-celled carcinoma, and who had anterior scalene nodes examined, were found to have metastases to these nodes. It should be noted that our series had an unusually low incidence of undifferentiated, small-celled carcinomas and a high incidence of gland-celled carcinomas. Inasmuch as these two types of carcinoma are those which are most likely to metastasize to scalene lymph nodes, and the undifferentiated-celled tumors usually occur at the pulmonary hilum while adenocarcinomas tend to be peripheral, the explanation for the higher incidence of positive nodes in association with peripherally located tumors in our series is probably due to the relatively small number of undifferentiated-celled carcinomas. If the incidence of the latter had been similar to that which has been reported by most investigators, it is likely that this difference between hilar and peripheral neoplasms would not have been significant.

Connar has emphasized the importance of removing sufficient tissue for an adequate anterior scalene lymph node biopsy and he regarded any specimen which contained less than 4 nodes as unsatisfactory. He found that reoperation in such instances usually disclosed lymph nodes which had been missed at the time of the first intervention. It has already been noted that an average of 5 lymph nodes per specimen was found in the patients with positive anterior scalene lymph node biopsies and 6 per specimen in the negative group. The smaller number in the former group was probably the result of less thorough sectioning of specimens which contained very large nodes or nodes which presented gross evidence of neoplasm. According to the criterion of Connar, 9 of the 22 negative specimens in our series would be regarded as unsatisfactory. If these are excluded from the analysis, the sensitivity of this diagnostic modality would be raised from 24 to 35 per cent, thus approaching that of bronchial biopsy (40 per cent).

Reports of cytologic smear studies illustrate considerable lack of uniformity of success in the detection of lung cancer. Shabart[11] found malignant cells in the smears of only 44 per cent of his patients with bronchogenic cancer, while others report positive results in over 95 per cent of their patients.[5] The

average sensitivity appears to be from 70 to 85 per cent. Malignant cells were found in the smears of sputum or bronchial aspirates of 69 per cent of the 42 patients in our series, and 88 per cent of the patients had smears which were interpreted as positive or suspicious for malignant cells.

Woolner and McDonald[15] found that 40 per cent of their patients who underwent thoracotomy for suspected lung cancer had positive cytologic smears as the only preoperative microscopic support of the clinical diagnosis. Gibbon and co-workers[6] reported a similar situation in 45 per cent of their preoperative patients. In our series, 26 per cent of the patients who had a positive cytologic diagnosis had negative bronchoscopic biopsies and negative anterior scalene lymph node biopsies. Only one patient who had a positive bronchial biopsy failed to disclose malignant cells in his sputum. It has been previously mentioned that all of the patients who had metastatic neoplasm in their anterior scalene lymph node biopsies also had positive cytologic smears.

Most observers have noted a decreased sensitivity in the cytodiagnosis of peripherally situated lung cancers.[6, 9, 13, 14] This observation has been confirmed in this study. Positive smears were not as frequently encountered in patients with peripheral tumors; nevertheless, only 3 of the 15 peripheral neoplasms were cytologically negative while all of these 15 patients had negative bronchoscopic examinations.

Although the least successful cytologic results were found in patients with carcinomas of the upper lobe (59 per cent), compared with patients who had lower lobe tumors (82 per cent) or tumors of the main-stem bronchi (100 per cent), these cytologic results in the case of upper lobe tumors represent a striking improvement over those obtained with bronchoscopic biopsy (11 per cent).

Combination of Diagnostic Methods.—It has been shown (Table II) that 100 per cent of bronchogenic carcinomas originating in a main-stem or lower lobe bronchus were diagnosed by one or more of the three diagnostic modalities under discussion. The utilization of this diagnostic triad permitted a preoperative, microscopic diagnosis of carcinoma in 90 per cent of the total group of 42 patients in this series. It is now the routine practice of the authors to use all three methods simultaneously, unless there is a specific contraindication to one or more of the procedures.

Operability.—Spjut and associates[13] found that a positive bronchoscopic biopsy indicated a 3:1 probability that the patient had an inoperable carcinoma. Others have not found a significant difference between the resectability of patients with positive bronchoscopic biopsies and those with negative bronchoscopic examinations.[1, 15] Our findings, which support those of Spjut, showed that lobectomy or pneumonectomy was performed in over 50 per cent of patients who had a negative bronchial biopsy and in only slightly over 25 per cent of those with a positive bronchial biopsy (Table IV).

The importance of anterior scalene lymph node biopsy in the determination of inoperability of pulmonary cancer is obvious. In this series it was found that a positive scalene node biopsy provided the only criterion of inoperability in 4 of the 7 patients who had positive biopsies.

The relationship between cytological findings and resectability is of interest. It has been claimed that there is no positive correlation between cytologic findings and resectability.[13] Nevertheless, in our series, the carcinomas of only 27.6 per cent of patients who had positive cytologic smears were resected, while 80 per cent of the group of patients who had negative cytologic smears underwent lobectomy or pneumonectomy. The cancers of 50 per cent of the patients who had suspicious smears were resected. These findings also raise some doubt concerning the generally accepted belief that early bronchogenic carcinomas are more capable of yielding positive smears than are more advanced neoplasms.[8] It should be noted that the incidence of resection in patients who had positive cytologic smears was almost identical to that of patients who had positive bronchoscopic examinations (27.5 per cent and 26.7 per cent, respectively).

CONCLUSIONS

1. A series of 42 histologically proved lung cancers disclosed a diagnostic sensitivity of 40 per cent by bronchoscopic biopsy, 24 per cent by anterior scalene lymph node biopsy, and 69 per cent (positive) or 88 per cent (positive or suspicious) by cytologic smear examination of sputum or bronchial aspirate.

2. Every carcinoma situated within a lower lobe or main-stem bronchus was detected by the use of this diagnostic triad.

3. Bronchoscopic biopsy was most successful in the detection of carcinomas of squamous-celled type and carcinomas in main-stem or lower lobe bronchi.

4. Anterior scalene lymph node biopsy was infrequently positive in patients with squamous-celled carcinomas or carcinomas of the lower lobes, while 50 per cent of patients who had undifferentiated-celled or gland-celled carcinomas and who had anterior scalene lymph node biopsies were found to have metastatic involvement of these nodes.

5. A positive anterior scalene lymph node biopsy provided the only criterion of inoperability in four of the seven patients who had positive biopsies.

6. Positive cytologic smears provided the only preoperative microscopic evidence of malignancy in 26 per cent of the patients and were successful in detecting a majority of bronchogenic carcinomas of all histologic types and locations, including those of upper lobes or situated in peripheral portions of the lung.

7. The resectability of carcinomas in patients who had positive bronchoscopic biopsy or positive cytologic smears was considerably less than that of those with negative results.

8. Careful cytologic study of smears of sputum or bronchial secretions proved to be a valuable diagnostic aid in suspected cancer of the lung in this series. Positive smears, properly evaluated in conjunction with clinical findings, have reliably served as presumptive positive evidence of cancer in the absence of other microscopic evidence and have permitted definitive thoracotomy. In only one instance was the cytologic diagnosis of neoplasm refuted histologically at operation.

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ESOPHAGEAL HIATAL HERNIA—ANATOMIC AND SURGICAL CONCEPTS, WITH SPECIAL REFERENCE TO THE EXPERIMENTAL USE OF AN IVALON PROSTHESIS IN THE REPAIR

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SEVERAL excellent surgical techniques[1, 5, 7] have been described for the repair of esophageal hiatal hernias. Most of these have given very satisfactory results, with a low rate of recurrence. There still remains an occasional case, however, in which present-day methods result in failure. It is in this small but important group of cases that the use of a prosthesis to strengthen the standard repair may become advantageous.

ANATOMIC CONSIDERATIONS

There are three salient anatomic structures that must be dealt with in the repair of the esophageal hiatal hernia: the muscular esophageal hiatus in the diaphragm, the fibrous phrenoesophageal ligament, and the serous peritoneal "sac" of the hernia. Each of these will be discussed separately, for it is felt that good repair of a hiatal hernia is dependent on a precise understanding of each of the three structures, as well as of their anatomic interrelationships.

Esophageal Hiatus.—The esophageal hiatus of the diaphragm is composed of two muscular crura which surround the lower part of the esophagus and have the shape of a teardrop, with the point of the drop being posterior. Allison,[1] Carey and Hollinshead,[2, 3] and others have pointed out that this muscular collar is almost always derived in its entirety from a splitting of the right crus of the diaphragm. Occasionally, the left crus may add a few muscle fibers to the formation of the hiatus, but this usually is an insignificant amount. The esophageal hiatus is the strongest anteriorly, for at that point there is a decussation of muscle fibers from both crura, forming a muscular sling or raphe that is almost unyielding. The central tendon of the diaphragm also is in close proximity anteriorly, further preventing enlargement of the hiatus in this direction. Lateral widening of the hiatus can occur, associated with hiatus hernia, but at the sides the muscular fibers of the hiatus are directed perpendicularly, thus strengthening it against gross lateral expansion. Posteriorly, the hiatus is virtually unprotected, and is easily split and enlarged.[2, 3] On the basis of these anatomic facts it is understandable, then, that a logical and important part of the repair of a hiatal hernia should be accurate approximation of the muscular fibers of the hiatus in its weak posterior portion.

Phrenoesophageal Ligament.—The phrenoesophageal ligament or membrane is a continuation of the fascia lining the inferior diaphragmatic surface as it passes through the esophageal hiatus to become attached to the fascia propria of the esophagus.[2, 3] Actually, this

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"ligament" is composed of a continuation of the same transversalis fascia which is so important in the pathogenesis of direct inguinal hernia. It supports the esophagus as that structure passes through the hiatus, and it helps to prevent displacement of the esophagogastric junction above the level of the diaphragm, thus contributing to prevention of the sliding type of hiatal hernia. When this ligament becomes weakened and stretched, it allows the esophagogastric junction to become displaced superiorly into the mediastinum, with subsequent bulging of the stomach through the muscular hiatus. The pathologic aspects here are much the same as those in direct inguinal hernia, in which the peritoneal sac with its contents bulges through a stretched and weakened transversalis fascia within Hesselbach's triangle. In esophageal hiatal hernia, just as in direct inguinal hernia, reconstruction and repair of this layer, whenever possible, should be carried out.

Peritoneal Sac.—The peritoneal sac in sliding hiatal hernia is formed by a redundant portion of peritoneum which is pushed ahead of the stomach or other viscus as it ascends into the posterior inferior mediastinum. The location of this peritoneal sac is almost always on the anterolateral aspect of the upper portion of the stomach and lower part of the esophagus. It never completely surrounds the hernial contents. The wall of the stomach usually is part of the hernial sac, and hence the derivation of the anatomically correct term, "sliding" hiatal hernia. Although the hernial sac plays no integral part in the formation of a hiatal hernia, it nevertheless represents a structure that must be dealt with in the repair. Either it can be excised in its entirety if small, or it can be incised along the margin of the hiatus, leaving its fundus in the posterior inferior mediastinum. Some authors advise plication of the sac, while others feel that its cut edge can be used to strengthen the hernial repair by sewing it to the diaphragm.

STANDARD SURGICAL REPAIR

Adequate exposure of the three essential anatomic components which require correction in the presence of esophageal hiatal hernia can be obtained by either a transabdominal or a transthoracic approach. If a concomitant intra-abdominal pathologic process, such as cholecystic disease, is present, the approach should be abdominal, so that both pathologic entities may be corrected through one incision. In such a case, a long upper midline or transverse upper abdominal incision is preferred. The transthoracic approach through the bed of the resected eighth rib or the eighth interspace is equally good, and perhaps gives even better exposure of the esophageal hiatus than does the abdominal approach. The repair to be described is similar to that of Allison,[1] and can be accomplished adequately from either the thorax or the abdomen, the only difference being the methods of exposure of the important anatomic structures.

The three essential structures of a sliding hiatal hernia are handled as follows. The peritoneal sac of the hernia is incised, with no attempt at complete excision unless the sac is very small. No definite attempt is made to use a portion of the sac in the hernial repair. Careful and accurate dissection of the muscular crura forming the hiatus is carried out. Interrupted sutures of heavy silk are used to bring these two crura together posterior to the esophagus in such a way that there is no residual constriction of the esophagus. If the surgeon can slide his index finger tip freely along the esophagus through the hiatus after the repair, an adequate lumen usually is assured.

The phrenoesophageal ligament is not always easily identified. If it can be found, the portion of this ligament which remains attached to the esophagus

is that used in the repair. It is attached to the repaired muscular hiatal opening in the diaphragm by interrupted sutures of silk. No attempt is made to fix the fundus of the stomach to the diaphragm. Closure of the abdomen or the thorax is then carried out in a layer-by-layer manner.

INVESTIGATIVE STUDIES

Several basic anatomic characteristics of the esophageal hiatus and its contents make repair of a hernia through this orifice of the abdomen basically less secure than the repair of a hernia in the inguinal or umbilical region. For one thing, the diaphragm is a dynamic and constantly moving structure, which means that considerable tension and to-and-fro movement involve any hernial repair at the hiatus. Furthermore, it is necessary to place sutures through muscle only, since there is no muscular fascia at the hiatus in which the emplacement of sutures would be effective. This means that muscle is coapted to muscle, a situation that does not provide maximal tensile strength in healing. Finally, although there is much reference to the phrenoesophageal ligament in the literature, it frequently is not such a definite structure as has been portrayed, and when the hernia is large it may be frayed out or even absent.

With these facts in mind, a technique was devised for the incorporation of a prosthesis in the repair, in an attempt to strengthen some of the basic anatomic weaknesses described. Polyvinyl-formol (surgical Ivalon) sponge* was chosen as the prosthetic material of choice for two reasons. First, it has been shown to have the remarkale quality of being physically and chemically inert in regard to surrounding tissues, and causes minimal "foreign-body" reaction. Second, it acts as an excellent scaffolding for the ingrowth of fibrous tissue without inciting a "reactive hyperplasia" of such tissue.[4, 6]

A C-shaped piece of Ivalon ¼ inch thick and of the noncompressed variety was used as the prosthesis. It was hoped that when this was placed around the hiatus with the terminal part of the esophagus in the center of the C, it would add considerably to the strength of the posterior hiatal repair without completely surrounding and constricting the esophagus. It was anticipated that such a prosthesis would serve as a phrenoesophageal ligament, so to speak, of particular benefit when this ligament was either very weak and markedly attenuated or when the hernia was so large in size that the posterior crural repair was inherently weak.

Experimental Surgical Technique.—Large esophageal hiatal hernias were created in dogs which were under the influence of intravenous pentobarbital sodium anesthesia produced through an endotracheal tube and with an artificial breathing device. A long thoracoabdominal incision was made on the left, through the eighth interspace, so that the costal arch was severed and excellent exposure of the diaphragm was provided from both the thoracic and abdominal sides. The mediastinal pleura was opened and the esophagus was mobilized. The phrenoesophageal ligament, which is well developed in the dog, was then

*Polyvinyl-formol (surgical Ivalon) sponge used in this study was supplied by the manufacturers, Simoniz, Inc., Chicago, Ill., and is available from Clay-Adams, Inc., New York, N. Y.

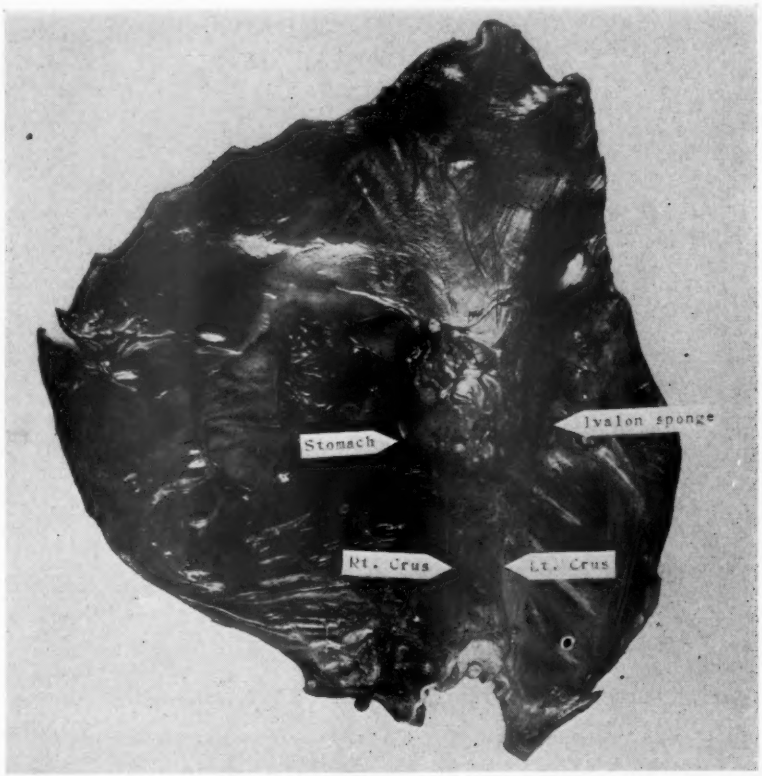

Fig. 1.—Repaired esophageal hiatal hernia in a dog 6 weeks after the corrective procedure Note minimal fibrous infiltration of Ivalon prosthesis.

Fig. 2.—Sections of Ivalon prosthesis 6 weeks after the surgical repair of esophageal hiatal hernia. Note infiltration of fibrous tissue into interstices of the sponge (hematoxylin and eosin; ×100).

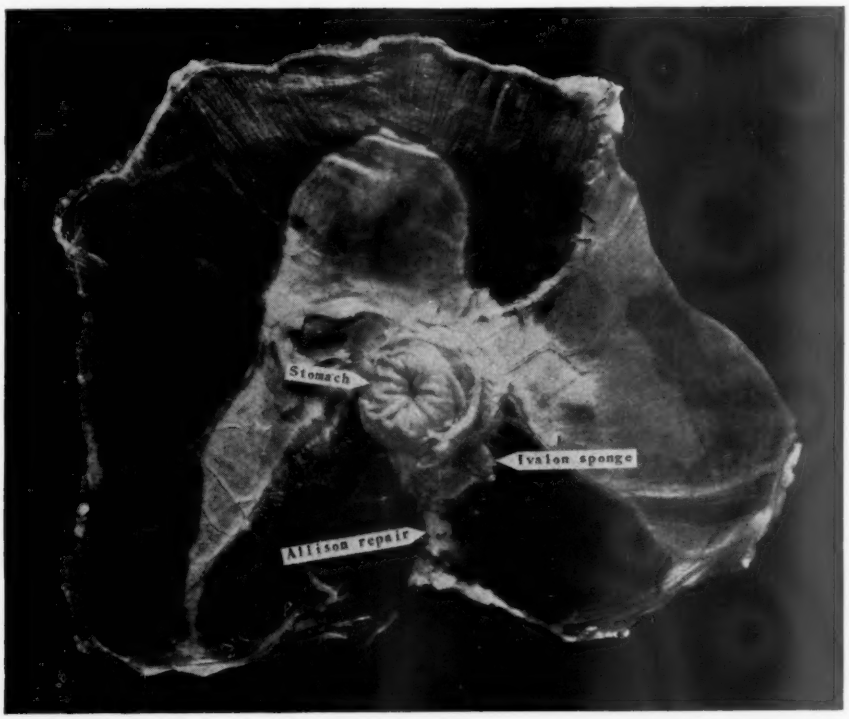

Fig. 3.—Repaired esophageal hiatal hernia in a dog 6 months after corrective operation. Note increase in fibrous encasement of the Ivalon prosthesis.

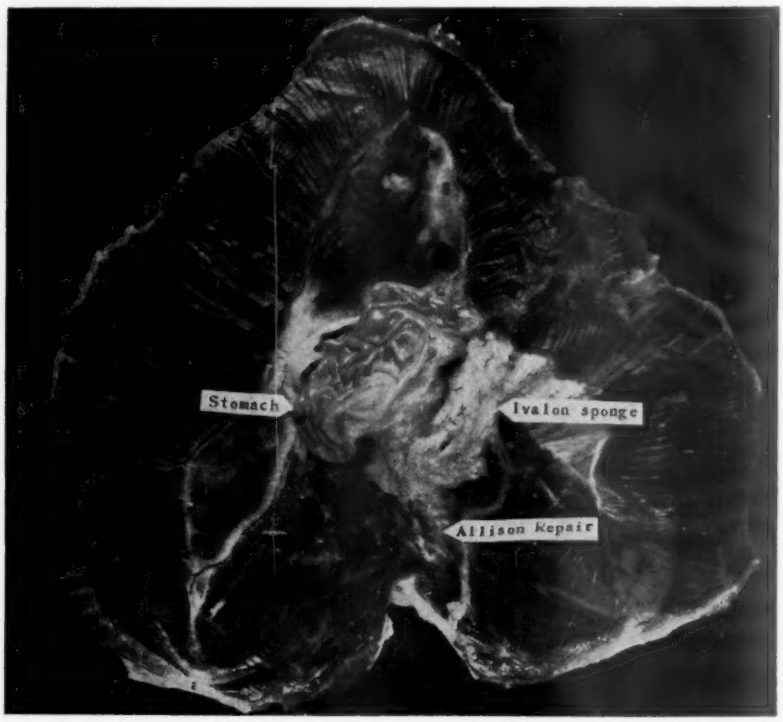

Fig. 4.—Repaired esophageal hiatal hernia in a dog 9 months after the corrective operation. Note how Ivalon sponge appears to strengthen posterior crural repair.

completely severed and the muscular hiatus was manually dilated until almost the entire stomach could easily be pushed through the opening with no difficulty. The repair was done through the abdominal approach. This approach was used in all cases for purposes of standardization, although certainly the technique could be used easily through the thorax alone. The muscular crura

Fig. 5.

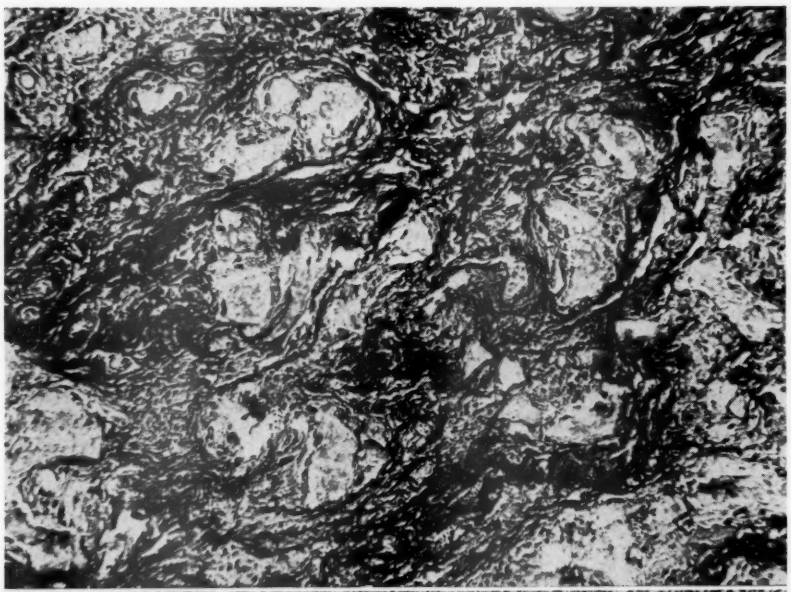

Fig. 6.

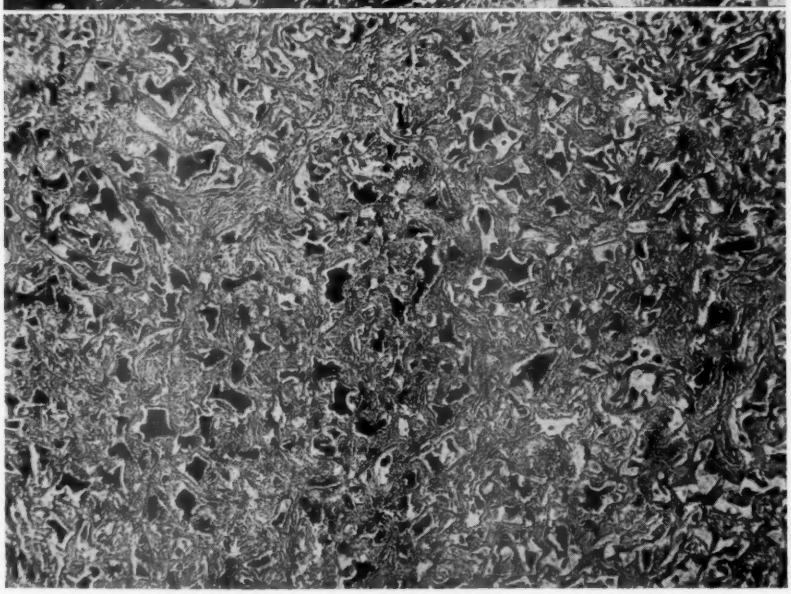

Fig. 5.—Section of Ivalon prosthesis 6 months after the corrective operation; tissue stained with elastic van Gieson stain. Note increase in fibrous infiltration over that evident in Fig. 2 (×100).

Fig. 6.—Section of Ivalon prosthesis 9 months after surgical correction of esophageal hiatal hernia. Additional fibrous infiltration into the interstices of the sponge is noted (×30).

of the hiatus were mobilized and posterior repair was effected with the use of interrupted sutures of silk. This constituted repair of the defect in the diaphragm.

A C-shaped piece of Ivalon was then fashioned so that the center of the C fitted loosely around the esophagus. This Ivalon prosthesis was sewed into place around the esophagus, with the open portion of the C to the right side of the abdomen. An inner row of fine silk interrupted sutures was used to attach the Ivalon prosthesis loosely to the fascia propria of the esophagus, and another layer was used to attach the prosthesis in its outer portion to the muscular diaphragm. Heavy suture material and tight coaptation of the prosthesis to the esophagus deliberately were not attempted, since it was desired to avoid tight coaptation which might favor stricture of this organ. No attempt was made to repair the cut phrenoesophageal ligament. Closure was carried out in a layer-by-layer manner, without establishment of drainage.

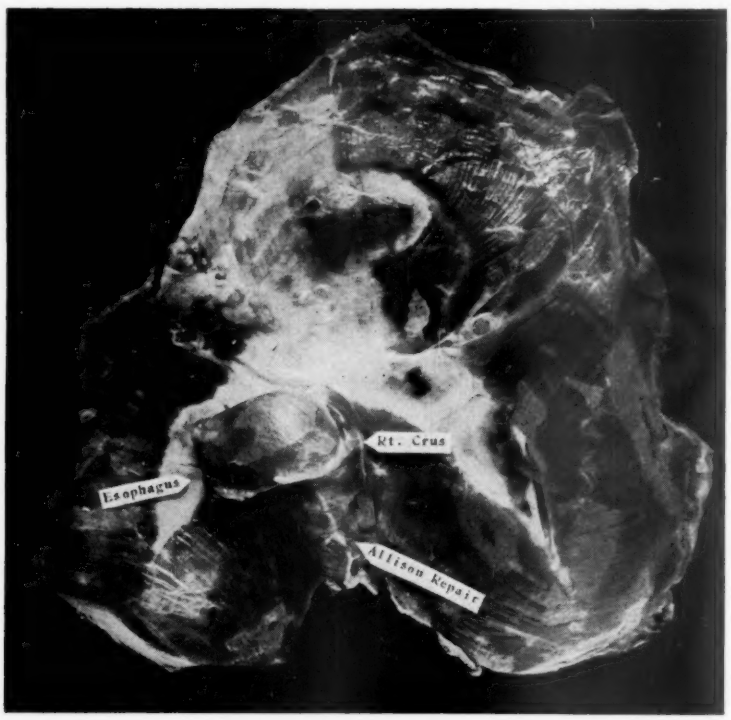

Fig. 7.—Thoracic view, 5 months after operation, of esophageal hiatal hernia that was repaired from the abdominal approach. Note the lack of reaction on the thoracic side to the repair below.

Results.—The creation of a sliding type of hiatal hernia and concomitant repair by means of a posterior crural suture and insertion of a C-shaped Ivalon prosthesis was carried out in 10 mongrel dogs. One of these dogs died in the early postoperative period (20 days), of esophageal obstruction. No attempt was made to dilate the esophagus of this dog. Post-mortem examination showed that the posterior crural repair enclosed the esophagus too snugly. No constriction was associated in any way with the Ivalon prosthesis.

The remaining nine dogs survived the operation uneventfully and in none of them were there any signs of esophageal obstruction. These dogs were killed at intervals varying from 6 weeks to 9 months from the time of surgery. Post-mortem examination was carried out in all instances, and esophageal obstruction was not found in any case except the one previously mentioned. The hernial repair likewise was intact in all nine, and there was no suggestion of a recurrence. Friable adhesions in the area of repair were common, as might be expected; however, in none of the dogs was there a granulomatous type of reaction around the Ivalon prosthesis. All specimens were carefully inspected for evidence of esophagitis and esophageal dilatation, but neither was found. It was noted that the fibrous ingrowth into the Ivalon was, in general, related to the time elapsing since operation. The dog killed at 6 weeks exhibited some gross invasion by fibrous tissue (Fig. 1), and the microscopic sections made at the esophagogastric junction also contained fibrous tissue in the interstices of the Ivalon (Fig. 2). In specimens examined at 6 and 9 months there was progressive gross encasement of the Ivalon with fibrous tissue, and this could be shown microscopically also (Figs. 3, 4, 5, and 6). In no instance was this fibrous reaction found to be in any way constricting the esophagus or obstructing it. All specimens were tested vigorously at the time of post-mortem examination to determine if the stomach could be displaced into the thorax. In all nine cases the repaired hiatus and Ivalon prosthesis exhibited almost unyielding strength. Fig. 7 shows the diaphragm of a dog killed 5 months after creation of a hiatal hernia and subsequent repair. This demonstrates the lack of reaction on the thoracic side of the diaphragm to the repair below. Certainly, although the thoracic approach was not used in this series, indications are that the Ivalon prosthesis would be just as efficacious and perhaps even more easily applied there, since exposure is more easily attained.

COMMENT AND CONCLUSIONS

Excellent results may be expected to follow the repair of an esophageal hiatal hernia if adequate attention is given to reconstruction of the basic anatomic abnormalities. Only occasionally will the size of the hernia or the anatomic alterations be such that the standard form of repair will be followed by recurrence. In the small group of patients who have marked enlargement and distortion of the esophageal muscular hiatus and associated weakening or absence of the phrenoesophageal ligament, it may be well to consider the use of a prosthesis to strengthen the posterior crural repair and to serve as the phrenoesophageal ligament. A C-shaped piece of Ivalon (polyvinyl) sponge has been found satisfactory in the dog for such a purpose. Experimental evidence is presented of the efficacy of this material in nine dogs in which large hiatal hernias were created and then repaired by means of an Ivalon prosthesis. In no case was esophageal obstruction from the Ivalon a problem, nor was reactive hyperplasia of the fibrous tissue infiltrating the Ivalon seen. There was no evidence of recurrence of the hernia 6 weeks to 9 months after the corrective procedure, and in all cases the esophageal hiatus exhibited great tensile

strength. On the basis of these data it is suggested that an Ivalon prosthesis might be useful as an adjunct to the standard posterior crural repair of some esophageal hiatal hernias of human beings.

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A FLEXIBLE AORTIC BIFURCATION GRAFT OF CHEMICALLY TREATED NYLON

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PREVIOUS reports from this department[1, 2] have outlined the development of straight tubes of braided nylon which, when chemically treated with formic acid and crimped, make a satisfactory flexible graft for arterial replacement. These tubes can be made in many sizes and lengths and have the following advantages. They are readily available and easily sutured to host arteries after heat-sealing the frayable ends of the tube. Bleeding through the pores is not a serious problem, and the crimping of the tube prevents wrinkling or kinking which could cause obstruction and thrombosis. It has been found both experimentally and clinically that the incidence of thrombosis is low even in very long tubes, as long as 20 inches. What the long-term results will be one year or more after insertion is not yet determined but the early experience up to 9 months has been excellent with no incidence of late thrombosis.

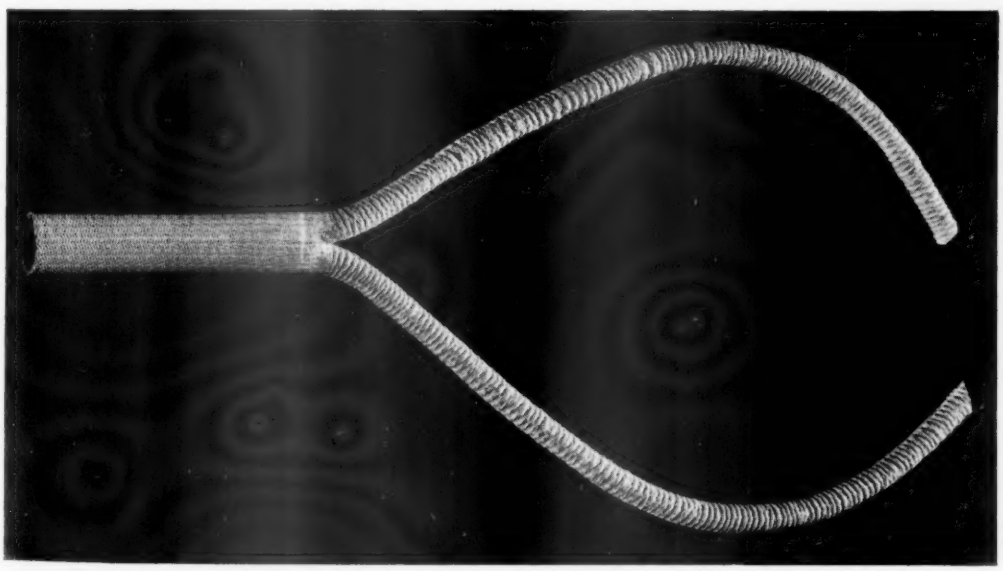

Fig. 1.—Braided nylon bifurcation graft chemically treated with formic acid to make a non-collapsible tube.

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A major problem in this field has been the development of a satisfactory prefabricated graft to replace or to by-pass the aortic bifurcation. It has been adequately demonstrated that it is possible to tailor and to fashion at the operating table "Y"-shaped tubes of various synthetic fabrics and to suture these to the patient's vessels successfully. Sanger[4] has developed a prefabricated "Y" tube of knitted Orlon which has been successfully used to replace the aortic bifurcation in aneurysms. Another "Y" tube of Dacron has been developed by Julian and Deterling[3] for the same purpose. If the porosity

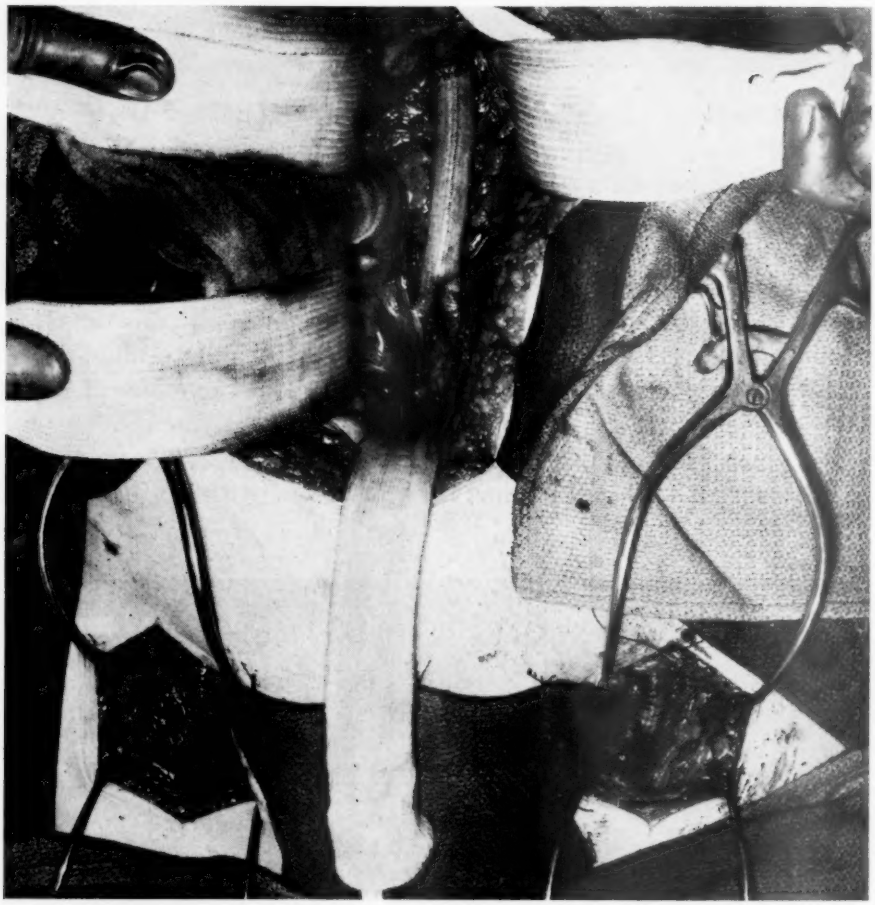

Fig. 2A.—Aortafemoral by-pass graft sutured to the stump of the aorta just below the renal arteries and to the side of the superficial femoral arteries through separate groin incisions.

of the tubes is low enough, bleeding is no problem with these prefabricated grafts and they are easy to suture because of the absence of cuffs or seams at the end which made technical suturing of the tailored grafts difficult. The one disadvantage of most of the tailored, as well as prefabricated, grafts has been the collapsibility of the cloth and the possibility of wrinkling or kinking on flexion of the tube. Our major efforts have therefore been directed toward developing a prefabricated "Y" tube with a straight aortic segment that was not flexible, but with iliac limbs which were noncollapsible and flexible

enough to allow end-to-side anastomosis to the iliac or femoral vessels below and to allow the patient to flex the thigh without the development of wrinkle obstruction. Early in our experience it was learned that it would be necessary to make this a braided tube since woven or knitted tubes cannot be crimped to allow the flexibility desired. A number of braiding problems were encountered in the development of a satisfactory "Y" tube with a tight enough braid so that the tube would not be excessively porous, even after chemical treatment. These problems were finally solved and it was found that the most satisfactory yarn size for the production of these "Y" tubes was 210

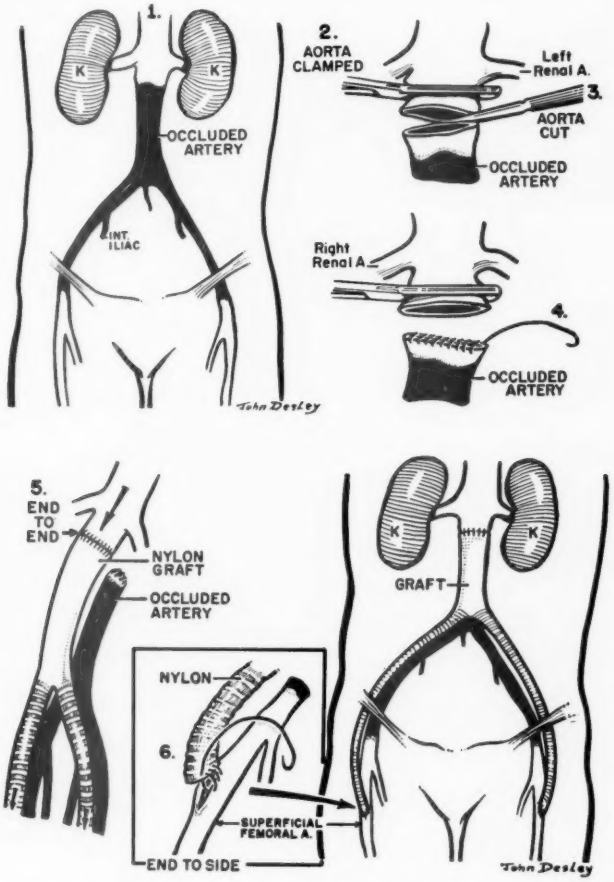

Fig. 2*B*.—Sketch of technique employed in the above operation.

denier nylon yarn, which is similar to that used in the straight tubular grafts. The final product has a 5/8 inch internal diameter straight aortic segment which is 5 inches long, while the crimped limbs are 3/8 inch in internal diameter and 12 inches long (Fig. 1). The 2 small holes on either side of the crotch are sealed with nylon-formic acid glue. This graft is treated with an ether solution of silicone to decrease blood loss through the pores.*

*Commercially available through C. R. Bard, Inc., Summit, N. J.

TECHNIQUES OF INSERTION

The "Y" graft is autoclaved for 20 minutes and can be resterilized several times without harm. At the operating table the graft is massaged and stretched to soften the crimps and, after exposure of the points of anastomosis, it is cut the proper length. This length is such that the graft should be very slightly under tension after completion of all anastomoses. Before insertion we have found it wise, although not absolutely necessary, to preclot the pores of the graft by withdrawing 50 c.c. of blood from the patient's arteries using a 50 c.c. syringe and No. 22 needle, injecting this into a small metal basin, and allowing the outer surface of the entire graft to come in contact with blood. By the time the anastomoses are completed there is almost complete closure of all the pores of the graft and very little blood will be lost

Fig. 3.—Photograph of an aortoiliac by-pass graft for bifurcation obstruction. Both limbs were sutured end-to-side to the external iliac vessels, the left limb being tunneled under the sigmoid mesentery.

through the walls. After the graft is cut, both ends of the tube are heat-sealed with a hot cautery as previously described to prevent any tendency toward fraying of the tube. The large end of the "Y" tube is then sutured to the end of the aorta at the point of division using a continuous over-and-over suture of 3-0 arterial silk interrupted twice. It is possible to suture the lower ends of the "Y" tube to the iliac, femoral, or popliteal vessels, either in an end-to-end or end-to-side fashion. End-to-end suture would be difficult if the distal vessel were smaller than the relatively large size of the limbs of the graft. Therefore, in most cases, even in the presence of aneurysm we have preferred the use of the end-to-side anastomosis of the iliac limbs of the graft

to the patient's distal vessels. This has several advantages. It gives a large anastomosis which is likely to thrombose and allows one to by-pass a long obstruction with minimal interruption of collateral blood flow around this obstruction. At the present time, 4 of these bifurcations have been used to by-pass aortoiliac obstructions. One by-pass (Fig. 2) extended from the aorta just below the renal vessels down to the superficial femoral arteries in the thigh. This was accomplished through separate incisions in the upper thigh to dissect out the patent superficial femoral vessels. This patient developed strong pedal pulses on both sides after operation and his claudication has disappeared. Three other aortoiliac obstructions were by-passed in a similar fashion, except that the operation was done entirely through the abdomen and the iliac limbs of the graft were sutured to the patient's external iliac vessels just inside the femoral canal (Fig. 3). All have developed strong pedal pulses and symptoms have disappeared. One ruptured abdominal aortic aneurysm has been replaced by this graft, but this patient succumbed to the loss of blood which she had suffered from a rupture of her aneurysm prior to emergency surgery. The graft sutured easily to her vessels and there were no technical problems resulting from the graft itself. In this case the 2 distal anastomoses were end-to-end to the relatively large common iliac arteries.

Heparin is introduced into the patient's distal vessels before application of the occlusive clamps using only about 10 mg. of sodium heparin in an effort to prevent thrombosis peripherally during the period of stasis. We have not used heparin postoperatively.

DISCUSSION

The chief advantage of this chemically treated bifurcation graft over tailored fabric "Y" tubes is its noncollapsible and nonkinking characteristic. The very valuable by-pass technique has been difficult to accomplish with flat collapsible tubes because of the likelihood of wrinkle obstruction. Whether nylon has any long-term superiority over other synthetic fibers is not yet known, but its reaction in animals and man has been studied far more than other fibers. It certainly appears durable and inert, and further chemical modifications should allow grafts to have all the favorable characteristics of homografts without the unfavorable factors of difficult procurement and occasional degeneration.

SUMMARY

Four cases of aortic bifurcation obstruction from arteriosclerosis have been successfully by-passed with a new braided "Y" tube of nylon chemically treated with formic acid. One ruptured abdominal aortic aneurysm was replaced with this graft but death followed from pre-existing shock. This prefabricated graft is easily sterilized and sutured, has a low incidence of early thrombosis, and has the advantage of having limbs that are flexible without kinking.

We wish to acknowledge the important contribution of Mr. Norman C. Jeckel of U. S. Catheter & Instrument Corporation, Glens Falls, N. Y., in developing a special braiding machine for producing the braided "Y" tube.

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IVALON SPONGE (POLYVINYL ALCOHOL) AS A BLOOD VESSEL SUBSTITUTE (FAILURE IN EXPERIMENTAL ANIMALS)

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IN THE continued search for a satisfactory substitute for segments of blood vessels lost due to disease, trauma, or congenital deformities, many synthetic materials have been tried with varying results. Ivalon sponge, or polyvinyl alcohol, was advocated by Shumway, Gliedman, and Lewis,[6] and later satisfactory results in human beings were reported by Cooley, Mahaffey, and DeBakey.[1] Theoretically, the material has several advantages. It can be molded into any shape, making a variety of branches possible. After molding, the graft has the shape and consistency of the normal blood vessel, making it technically easier to insert. The grafts are also elastic.

This paper is a report of the results obtained in 25 dogs with molded grafts of Ivalon sponge* in the thoracic and abdominal aortas, carotid and femoral arteries.

METHOD

Blocks of Ivalon sponge were frozen and slices 3 mm. in thickness were cut with a meat slicer. These were thoroughly washed in water to remove preservatives. The grafts were prepared in molds modified from those used by Shumway, Gliedman, and Lewis.[6] Two to three thicknesses of the wet sponge were wrapped around the inner rod and compressed to a thickness of 1 mm. The thoracic grafts were tapered for better fit. These were then placed in the autoclave for 30 minutes and heated at a temperature of 250° F. The molds were removed after cooling. The resulting grafts were uniform in thickness and smooth on the inside. There was an occasional defect in the grafts after compression, resulting from overlapping of the pores of the sponge. These were removed, or the grafts were discarded.

Adult mongrel dogs were used in the study. All the animals were anesthetized with intravenous sodium pentobarbital. Endotracheal tubes were inserted and oxygen was given by intermittent positive pressure with a pneophore valve.

The chest was entered through the fifth interspace. The proximal 6 inches of the descending thoracic aorta were freed, then a shunt of polyethylene tubing was placed between the left subclavian artery and the descending aorta during the period of occlusion. A segment of aorta 4 cm. in length was excised between Potts clamps and replaced by a tapered Ivalon sponge graft 6 cm. in

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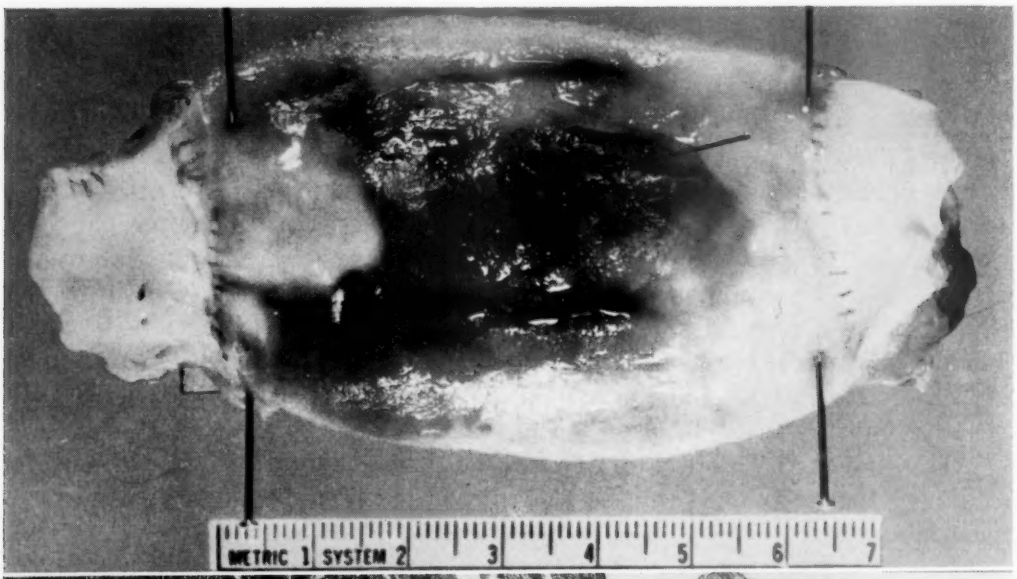
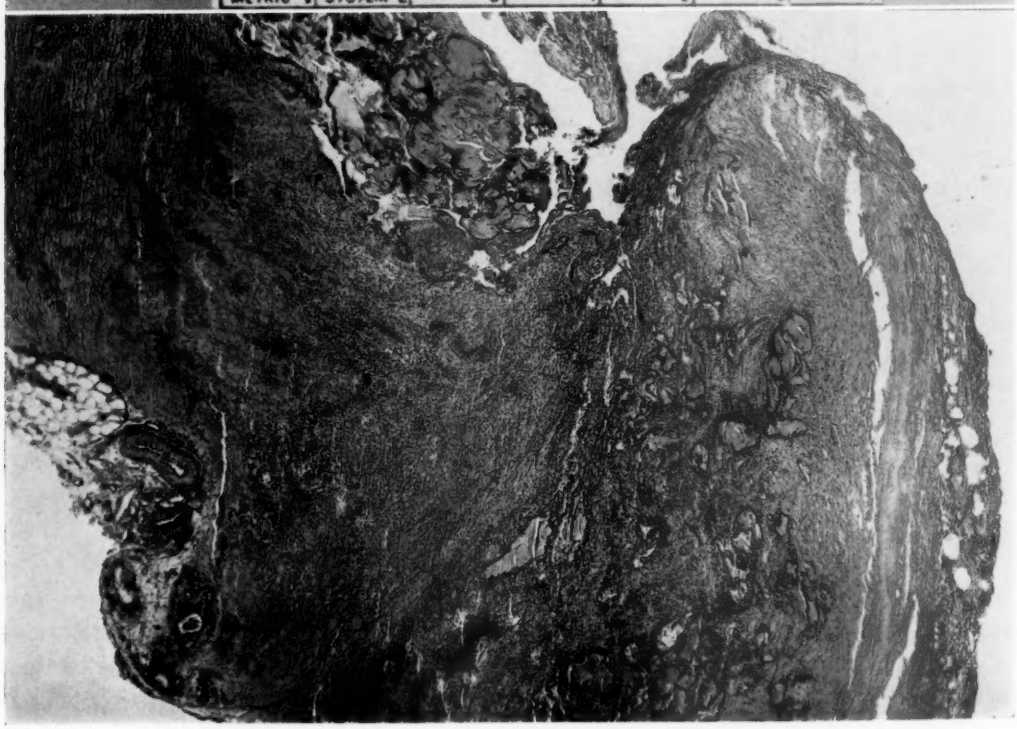
*Ivalon Surgical Sponge—Formulation M. Supplied by Clay Adams Company, 141 East 25th St., New York 10, N. Y.

length. Interrupted everting mattress sutures were placed at two corners and the anastomoses completed with a continuous over-and-over suture of No. 5-0 artery silk. The shunt was then removed, the defect in the aorta closed with a continuous over-and-over suture of No. 5-0 artery silk, and the subclavian artery ligated.

A.





B.

Fig. 1.—Dog No. 404. Rupture of thoracic Ivalon sponge graft at 98 days. *A*, Photograph of inner surface of graft. Arrow points to site of rupture. *B*, Photomicrograph of area of rupture showing breakthrough, degradated Ivalon sponge, and fibrous tissue.

The abdominal aorta was exposed through a midline incision and freed from the renal arteries to the trifurcation. A 4 cm. segment was removed between Potts clamps and replaced by a graft the same diameter and 5 to 6 cm. in length. The same suture technique was used in these as was used in inserting the thoracic grafts.

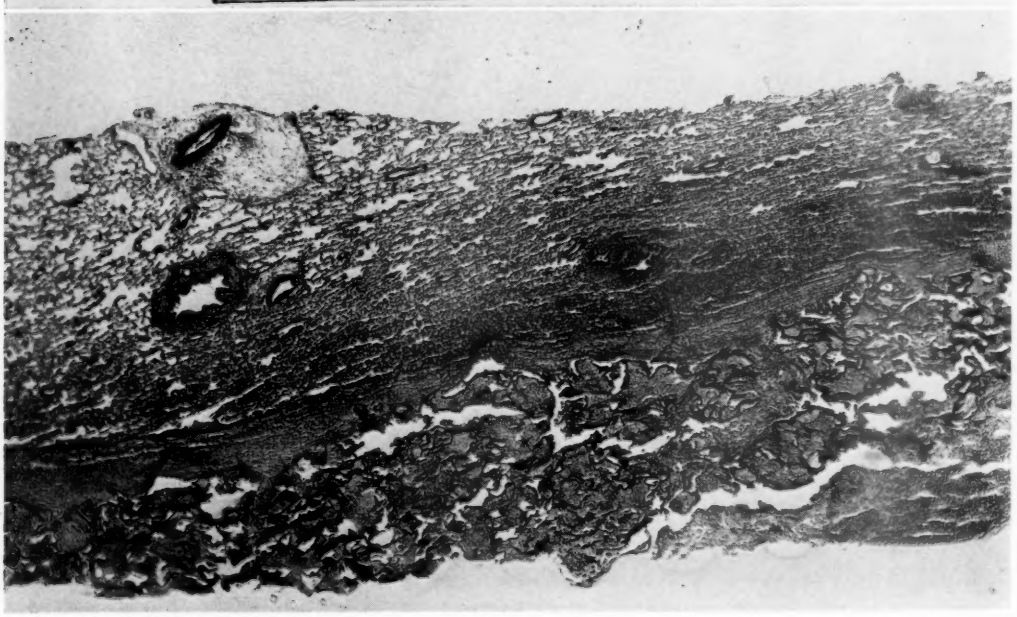
Fig. 2.—Dog No. 431. Ivalon sponge graft, thoracic aorta, 183 days. *A*, X-ray showing dilatation of graft. Silver clips mark extent of graft. (*B* and *C* on following page.)

The femoral and common carotid grafts were inserted into large animals only. A segment 3 cm. in length was excised and replaced by a graft 4 cm. in length and 4 mm. in diameter. Anastomoses were done in the same manner as above.

Postoperatively, all animals were given penicillin for one week. They were followed carefully for changes in pulses and by aortograms, then sacrificed at intervals up to six months. Complications precluded the necessity for

longer periods of observation. All grafts were studied grossly, photographed, and placed in 10 per cent formalin. Blocks were embedded in paraffin and sections stained with hematoxylin and eosin for histologic study.

B.



C.

Fig. 2 (Cont'd).

B, Photograph of inner surface of graft showing bare areas, thrombi, and separation of fibrous lining.

C, Photomicrograph of bare area. Note slough of inner part of sponge.

RESULTS

The grafts were technically easy to insert, because of a consistency similar to that of the proximal and distal artery. Following restoration of flow, there was minimal blood loss which stopped completely after one to three minutes. The walls of the grafts were elastic and pulsated well.

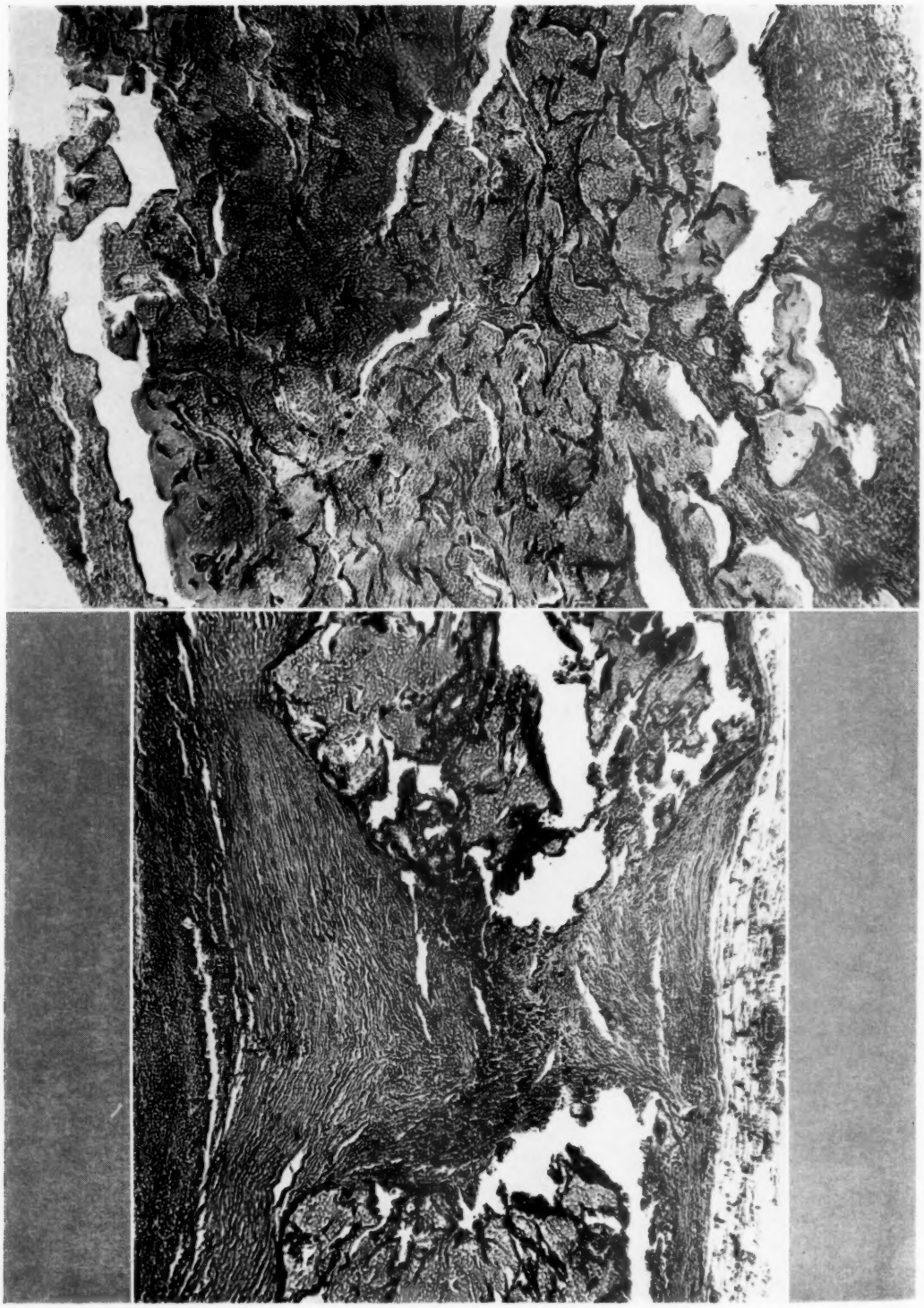
Thoracic.—Nine dogs composed this series.

The polyethylene shunts functioned satisfactorily during the period of occlusion of the thoracic aorta, none of the animals developing shock or paralysis. All the grafts remained patent during the period of observation (Table I). The Ivalon sponge early became hard and brittle, losing all its elasticity. One animal exsanguinated at 17 days postoperatively from rupture of the distal suture line. Two exsanguinated at 73 and 98 days postoperatively from aneurysm and rupture of the grafts (Fig. 1). All of the remaining six developed fusiform dilatations of their grafts which progressed during the six months' observation period (Fig. 2).

TABLE I. SUMMARY OF RESULTS WITH IVALON SPONGE GRAFTS IN THORACIC AND ABDOMINAL AORTAS

DOG NO.	LENGTH (CM.)	INTERNAL DIAMETER (MM.)	DAYS FOLLOW-UP	CAUSE OF DEATH	STATUS OF GRAFT
				Thoracic	
429	6	11	17	Hemorrhage	Rupture of suture line
434	6	12	73	Hemorrhage	Aneurysm and rupture
404	6	10	98	Hemorrhage	Aneurysm and rupture
498	5	12	48	Cardiac arrest following aortogram	Aneurysm
416	5	10	180	Sacrifice	Aneurysm—Bare areas in central portion
435	6	10	186	Sacrifice	Aneurysm—Bare areas in central portion
431	5	12	182	Sacrifice	Aneurysm—Bare areas
407	6	11	172	Sacrifice	Aneurysm—Bare areas
				Abdominal	
401	6	6	73	Sacrifice	Thrombosis at 21 days
423	5	6	56	Sacrifice	Thombosis at 16 days
502	5	6	21	Hydronephrosis	Patent; small thrombus in mid-portion
438	5	6	95	Sacrifice	Patent
430	5	6	143	Sacrifice	Patent; rigid
406	5	6	140	Sacrifice	Patent; rigid
315	5	6	183	Sacrifice	Aneurysm in mid-portion
424	6	6	195	Sacrifice	Patent; rigid
304	6	7	189	Sacrifice	Rupture of graft with dissecting aneurysm

A.



B.

Fig. 3.—Photomicrographs of walls of Ivalon sponge graft. *A*, 17 days. Ivalon sponge is intact and there is ingrowth of fibrous tissue. *B*, 180 days. Broken areas of sponge have been replaced by fibrous tissue.

The grafts became enclosed in a fibrous capsule which was adherent after three to four weeks. Fibrous ingrowth occurred through the interstices of the sponge. There was early a fibrin lining on the inner surface of the grafts. This was replaced by a smooth fibrous lining extending from the proximal and distal ends and areas in the central portion. Areas not covered by the fibrous lining were covered by a ragged, thick, fibrin layer with multiple thrombi. The new lining became broken and separated in the central portions of most of the grafts as a result of dilation from giving of the sponge. In the ones observed at four to six months there were bare areas where portions of the lining had apparently become detached (Fig. 2, *B* and *C*). Microscopically, there was evidence of widespread degeneration of the Ivalon and points of breakage replaced by fibrous tissue (Fig. 3).

A.

B.

Fig. 4.—Dog No. 315. Photographs showing aneurysm in proximal portion of graft, 180 days. *A*, Outer view. *B*, Graft opened.

Abdominal.—Nine grafts, 6 mm. and 7 mm. in diameter, were placed in the abdominal aorta (Table I). Two became occluded by thrombosis at 16 and 21 days. The remainder of the animals maintained good femoral pulses until the time of sacrifice. One examined at six months had an aneurysm in the central portion (Fig. 4). Another developed a dissecting aneurysm, the

Ivalon sponge becoming completely separated from the surrounding tissue by old blood (Fig. 5). The ingrowth of fibrous tissue was similar to that detected in the thoracic grafts.

Femoral and Carotid.—Seven grafts, 4 mm. in diameter, were placed in the femoral and common carotid arteries. Six of seven were unsuccessful due to occlusion from thrombosis during the first 14 days.

Fig. 5.—Dog No. 304. Dissecting aneurysm in Ivalon sponge graft, 188 days. Arrow points to site of rupture. Graft has been dissected by blood from surrounding fibrous tissue.

DISCUSSION

To function satisfactorily an artery graft must remain patent and its walls be sufficiently strong to prevent aneurysm or rupture. In the thoracic aorta of dogs occlusion from thrombosis did not occur with grafts of Ivalon sponge. Similar results have been obtained in 82 grafts of nylon, Dacron, Orlon, and Teflon.[2] As the diameter of the graft decreases, the results become less encouraging. The 25 per cent rate of occlusion of grafts in the abdominal aorta with Ivalon is as low or lower than that obtained with grafts of nylon, knitted Orlon, and Dacron.[2] The results in replacement of arteries less than 6 mm. in diameter with Ivalon sponge is the same as that noted with other materials, most of them becoming occluded by thrombosis.[2]

Following implantation a plastic prosthesis becomes enclosed in a fibrous capsule which adds support but which is not sufficient, particularly in the early stages, to prevent aneurysm or rupture. A synthetic material from which a graft is to be made must be strong enough to withstand the arterial pressure and sufficiently chemically inert to maintain this strength for a long period of time. Ivalon sponge is initially strong enough, but the results in

this study indicate that this strength is not maintained. It is somewhat unstable chemically, being attacked by strong or weak acid or alkali and is water-soluble.[3-5] This instability and solubility account for its rapid breakdown when subjected to the body fluids, resulting in a high incidence of aneurysm and rupture of grafts after a short period of time.

CONCLUSIONS

The advantages that Ivalon sponge presents as an artery substitute are overweighed by its disadvantages. Because of its early friability and breakdown with aneurysm and rupture, it is felt to be unsatisfactory for the replacement of blood vessels.

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LEFT INTERNAL MAMMARY ARTERIOCARDIOPEXY IN THE THERAPY OF CORONARY ARTERY INSUFFICIENCY

A Preliminary Experimental Report

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PREVIOUS publications from this laboratory have substantiated that the internal mammary artery can be successfully introduced and is capable of supplying arterial blood to an ischemic myocardium.[1, 9-12, 15-19] Numerous alterations in the implant technique have been and are being attempted in order to enhance the benefit which might be derived from the transplanted internal mammary artery in the surgical treatment of coronary artery insufficiency. In order to realize the fullest potential from this artery, attempts are being made to stimulate more branching from the vessel and increase the percentage of anastomoses with surrounding arterioles. Furthermore, repeated experimental trials with the standard internal mammary artery implant and its immediate modifications constantly yield a small percentage of arteries which angulate, thrombose, block, and therefore cannot function to their fullest capacity. Over the past 11 years, the large series of dogs studied in this laboratory has provided evidence that approximately 75 per cent of implanted internal mammary arteries form satisfactory communications with the coronary arterioles. The success of the implantation procedure seems to depend upon the proper technique of implantation and a receptive (ischemic) myocardium.[14, 19]

A detailed review of all previous investigation on the implantation of the internal mammary artery in the Experimental Surgical Laboratories of McGill University has been carried out. As a result, certain facts have become apparent which suggested modification of the standard internal mammary artery implant procedure, which are as follows:

Arborization of the Detached Internal Mammary Artery.—One group of experiments performed in 1948[11] was most instructive. The internal mammary artery was freed from the subclavian artery to the level of the sixth intercostal space, and the distal transected end was buried in a tunnel within the left ventricular myocardium in 39 dogs. At that time, it was not yet realized that the artery had to be implanted with a freely bleeding sixth intercostal branch, and all of these arteries on histologic section were found to be blocked within the myocardial tunnel. However, the most significant finding in 14 of the

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dogs which survived one month or more was that the internal mammary artery had spontaneously readhered to the chest wall in 10 of the 14 and had formed new intercostal arteries in most of the intercostal spaces. The tendency of a detached internal mammary artery to send out new arterial branches to surrounding structures has been observed repeatedly throughout the years with reference to fat pads, and on occasion to the surface of the myocardium.[4, 13, 14, 19]

Removal of the Epicardial Barrier.—Theoretically, the predominating factor which might prevent a detached internal mammary artery from acting in a similar manner on the surface of the heart by infiltrative arborization is the presence of the epicardial barrier. Experience in this laboratory with pericardial fat pad grafts confirms this hypothesis.[4, 18] It has been shown that careful removal of the epicardium results in a very high percentage of anastomoses between the vessels of the fat pad and the coronary vessels. In addition, the procedure of the pericardial fat pad graft has been used successfully at this center in the surgical treatment of human coronary artery insufficiency as a supplement to the standard internal mammary artery implant procedure.

Following the development of special instruments in this laboratory, the epicardium can be widely extirpated over the right and left ventricles of animal hearts producing little trauma to coronary vessels or underlying myocardium.[7]

Importance of Maintaining a Free Flow of Blood in the Transplanted Artery.—Allusion has been made previously in this report and other publications from this laboratory concerning the importance of implanting the internal mammary artery into the myocardial tunnel with a freely bleeding intercostal branch. Experience has proved that without a comparatively free flow of blood through the artery, the intimal and medial tunicae undergo hyperplasia with subsequent thrombosis and finally, blocking of the vessel. Angulation of the vessel as it enters the tunnel or when it leaves the chest wall produces somewhat the same result.

Having established that arborization might be expected from the internal mammary artery and that these branches could conceivably penetrate the ventricular myocardium if the epicardial barrier were properly removed, it was decided that tunneling into the myocardium may be superfluous. Furthermore, the flow of blood within the artery would be least disturbed if the entire internal mammary artery complex were to remain intact without ligation or transection. This principle was attempted in one of our earliest studies,[9] whereby the artery was freed from its bed in its central portion and inserted into a slit made in the wall of the left ventricle with a free pleural graft or sutures holding it in place. This artery was found to have its lumen completely occluded by fibrous tissue at the site of attachment in the left ventricular slit. The lumen of the artery proximal and distal to the site of transplantation remained patent.

Thus having reached this stage in both experimental and clinical experience, it was considered worthwhile to transfer the systemic vascular potential of the internal mammary artery to the surface of the ventricular myocardium as an uninterrupted loop where it might prove to function as an entirely new coronary artery.

TECHNIQUE OF INTERNAL MAMMARY ARTERY COMPLEX TRANSFER

Mongrel dogs were used in these experiments, anesthetized with Nembutal, positioned with the left thorax uppermost, and intubated with an endotracheal tube which was connected to an intermittent positive pressure breathing apparatus after the pleura had been entered.

The thorax was approached through the left sixth or seventh intercostal space. A 1 cm. width of parietal pleural and any underlying transversus thoracis muscle was removed from the entire area overlying the left internal mammary artery from the level of the left subclavian artery to the diaphragm. The internal mammary artery was then gently teased from its bed and all its branches were doubly ligated and separated. The fifth, sixth, and seventh intercostal branches were ligated at least 1 cm. from their origins and the ends of the ligatures were left long for purposes of attachment to the ventricular myocardium.

All available fat pads were separated from the anterior surface of the pericardium, allowing their mediastinal and diaphragmatic attachments to remain intact. The anterior portion of parietal pericardium was removed.

The freed loop of internal mammary artery fell into place over the ventricles and usually assumed a position of its own natural tendency on the anterior surface of the heart extending from the pulmonary conus to the apex of the left ventricle. After visually outlining this course, the epicardium was stripped along this entire path extending over right and left ventricles to the apex and decussating with the anterior descending ramus of the left coronary artery. A special instrument, previously alluded to in this report has been developed for this purpose.[7]

The long ends of ligatures on the intercostal branches were then loosely sutured to the epimyocardium on either side, attaching the artery to its previously determined bed on the bared myocardium without tension. The previously prepared mediastinal fat pad was then pulled over the transplanted internal mammary artery and attached loosely by three or four sutures to the epimyocardium approximately 2 cm. to the left of the transplanted vessel. Routine closure of the thorax was then completed after careful reinflation of the lung.

EXPERIMENTAL DATA AND RESULTS

In order to produce some degree of myocardial ischemia, DuPont PT-450 cellophane with or without dicetyl phosphate coating was wrapped around the origins of the anterior descending and circumflex branches of the left coronary artery at the time of the arteriocardiopexy. These substances have since been found inferior to a material which we are now using, called Ameroid which is a casein plastic, in producing gradual coronary artery occlusions.[2, 3, 5, 6]

Acute experiments were performed usually 2 months following the left internal mammary arteriocardiopexy, and the following significant results were noted.

There is an indication, as evidenced by the electrocardiographic tracings in Fig. 1, that sufficient amount of coronary artery occlusion was produced

by the cellophane and dicetyl phosphate in some instances. Serial standard limb lead tracings were taken while both ends of the implanted vessel were temporarily occluded with noncrushing clamps.

Following the acute experiment with ECG study, all of these dogs were sacrificed and the hearts were removed en masse together with the entire attached internal mammary artery complex and refrigerated for 24 hours to allow relaxation of muscular spasm due to rigor mortis. Following this, the hearts were thoroughly warmed in physiologic saline solution to a temperature of 44° C.

Some of the transplanted internal mammary arteries were injected with India ink to determine the presence of anastomoses from the arteriocardiopexy to coronary vessels. Microscopic sections (Fig. 2) reveal the presence of India ink which was injected into implanted vessels deep in the myocardial vasculature. Histologic evidence of anastomotic communications between transplanted artery and myocardium has been found in every specimen examined. Also, there is no evidence of angulation, thrombosis, or endarteritis with intimal or medial hyperplasia of significance in any specimen of this series examined 60 days after arteriocardiopexy.

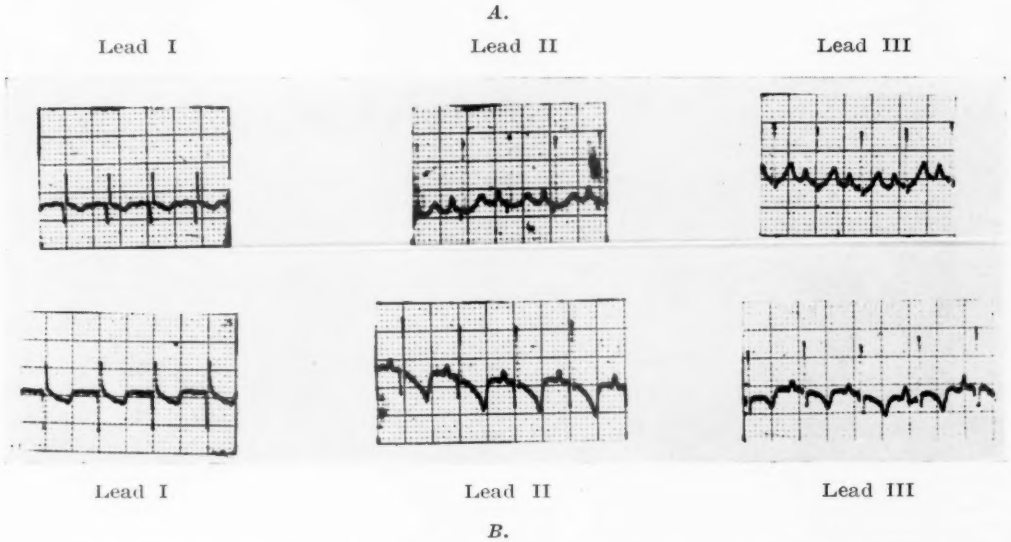

Fig. 1.—Photograph of standard ECG limb leads illustrating results of an acute experiment 60 days after internal mammary arteriocardiopexy. Tracings in *A* were taken prior to occlusion of the transplanted vessel as a control. Tracings in *B* were taken 15 minutes after the vessel had been clamped at either end. Note the slight elevation of ST segment in lead I and inversion of T waves in leads II and III.

Of the first series of animals subjected to this type of procedure, 5 have been sacrificed 60 days following the arteriocardiopexy, and one died with no abnormalities other than acute dehydration and malnutrition 30 days after operation in spite of adequate intravenous therapy.

Three of this group had the anterior descending and circumflex branches of the left coronary artery wrapped with DuPont PT-450 cellophane, while the other 3 had PT-450 cellophane wrapping dipped in a saturated solution of dicetyl phosphate in toluene. The cellophane strips were one-half inch wide and

doubly wrapped. Ischemic changes on ECG (see Fig. 1) were apparent in 2 dogs of this group when the implanted artery was occluded at either end. This coincided with a small amount of coronary artery narrowing at the site of the cellophane only in one and the combination of cellophane and dicetyl phosphate in the other. None of the wrapped coronary vessels in the remainder of this group were significantly narrowed upon examination by probing.

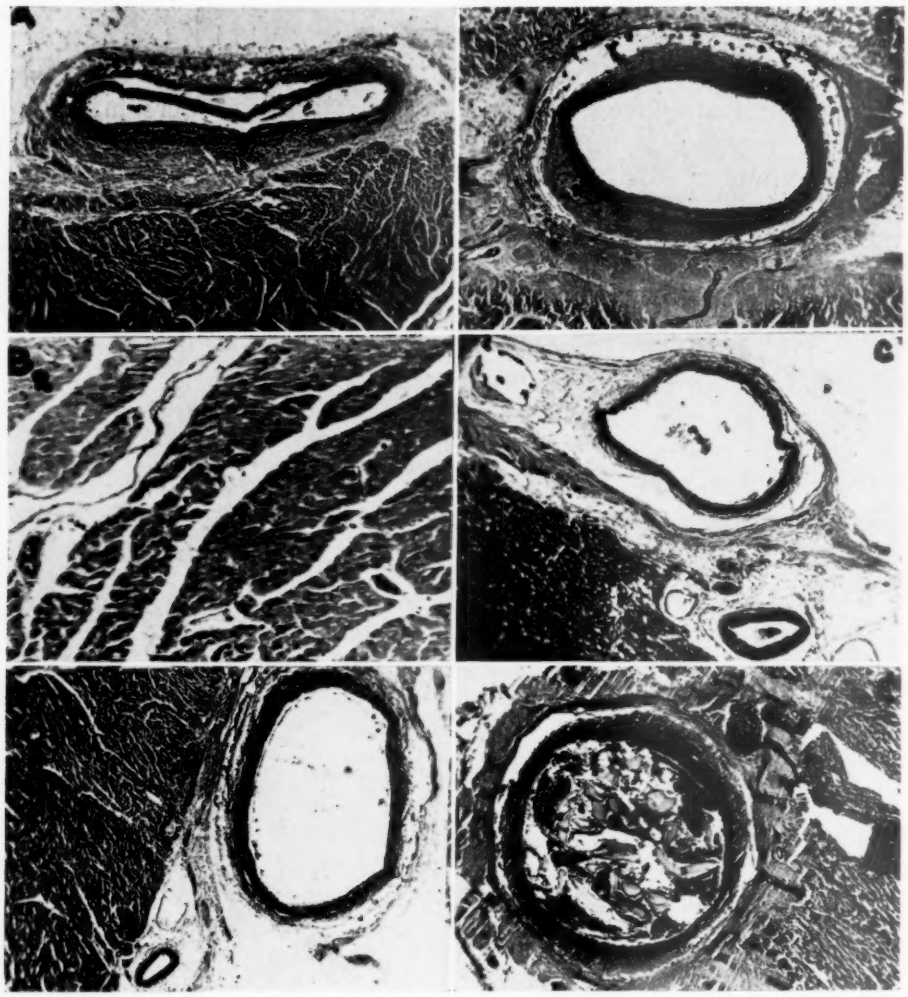

Fig. 2.—Photomicrographs of sections through the arteriocardiopexy and underlying myocardium of hearts in this series. The internal mammary arteries have been injected with India ink and/or Schlesinger solution.[8] Note the relative absence of intimal or medial hyperplasia in the grafts as well as the presence of ink in the anastomotic vessels and deeper in the myocardium (b_1 and b_2). a, Thirty-day-old implant (×25). b, Sixty-day-old implant (×25). b_2, Coronary vessels containing India ink in myocardium (×120). c, Sixty-day-old implant (×25). d, Sixty-day-old implant (×25). e, Sixty-day-old implant filled with Schlesinger mass (×25).

Five of the transplanted arteries were found to be patent in their entirety and carried a flow of normal saline at 100 mm. Hg pressure of 50 to 80 c.c. per minute. The sixth implanted vessel was blocked at its distal end on exit from the surface of the heart, but carried a flow of normal saline and India ink into the heart at 10 c.c. per minute at 100 mm. Hg pressure, part of which could be recovered at the left coronary ostium.

In the second series of dogs now in progress (which will be considered more adequately in a later publication) using casein plastic sleeves to gradually occlude the branches of the left coronary artery, one has died 30 days after internal mammary arteriocardiopexy of a pulmonary infection. This occurrence has availed us with an additional specimen for the present report. The heart was injected through the transplanted vessel with Schlesinger solution.[8] An x-ray of the injected heart (Fig. 3) revealed early branching from the implant and the presence of a small amount of Schlesinger mass in the anterior descending ramus of the left coronary artery. Schlesinger solution always penetrates to arterioles 40 micra in diameter, reaches about 50 per cent of the vessels 20 micra in diameter, and never injects vessels smaller than 10 micra in diameter.

The paucity of hyperplasia of both the medial and intimal tunicae of the transplanted arteries has been noted in every specimen examined to date.

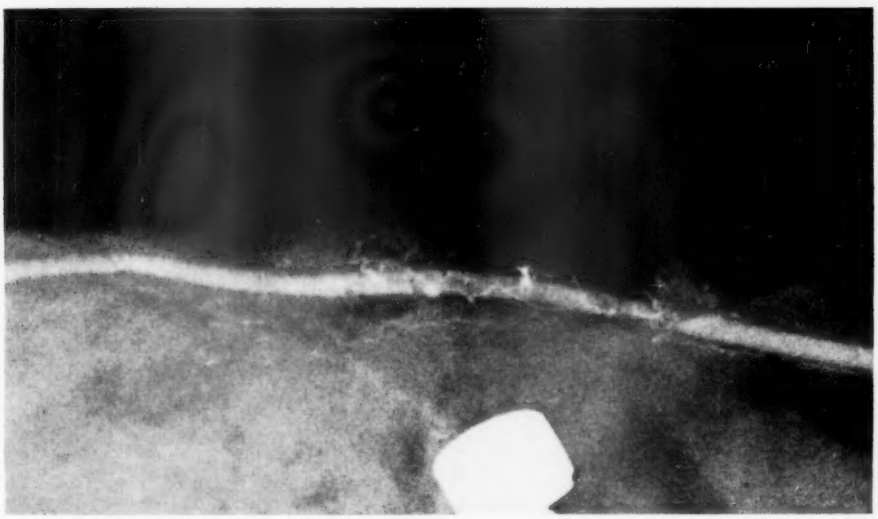

Fig. 3.—Radiograph of heart 30 days after internal mammary arteriocardiopexy. The transplanted vessel was injected with Schlesinger solution. Note the early branching from the vessel and the presence of a small amount of Schlesinger mass in the anterior descending ramus. (Casein plastic sleeves are on vessels of the left coronary artery.)

CONCLUSIONS

A review of experimental and clinical work done in this laboratory has furnished information concerning proliferative phenomena associated with the internal mammary artery which is now being investigated more thoroughly using casein plastic sleeves to produce a more realistic type of coronary artery insufficiency.[6]

Removal of the epicardial barrier appears to have enhanced adequate vascular union between proliferating branches of the transplanted vessel and those feeding blood to an ischemic myocardium.

An implanted internal mammary artery which previously blocked in some instances due to angulation, thrombosis, intimal proliferation, or dubious surgical technique now appears to remain fully patent and functional when allowed

to remain intact as a complex loop (arteriocardiopexy) and maintained in direct apposition to the bared myocardium. This work concerns a small series of dogs studied over a period of approximately 60 days after transplant.

It is emphasized that the procedure herein described is only one of numerous new techniques which are being attempted in this laboratory, and it remains strictly in the experimental stage.

We wish to express appreciation for skilled operating room management, technical assistance, and x-ray and photographic work to Mr. Paul Rouston. Histologic sections were prepared by Mr. Ray Jones, and Miss B. M. Macario typed and corrected the manuscript.

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THE USE OF TONGUE FLAPS IN HEAD AND NECK SURGERY

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THE tongue is the largest organ in the oral and pharyngeal cavities. Its position and composition make it ideal for use in the covering and closure of certain surgical wounds in the head and neck. These wounds are in relationship to the mandible, cheek, lower lip, pharynx, palate, and neck and are usually the result of extensive extirpations for the control of cancer. In many instances their management may be accomplished without mobilization of the tongue. There are situations, however, where the tongue may be used as a temporary dressing or as an essential dressing facilitating a one-stage operation, shortening morbidity, and improving the security of the technique. These techniques do not seriously compromise the functions of swallowing and talking as they relate to the tongue. Neither do they affect esthetics.

INDICATIONS

Tongue to Lateral Cheek.—Extensive cancer of the floor of the mouth, tongue and/or mandible, and lateral neck requiring a radical resection of all of these composite structures creates a void from the oral cavity to the clavicle. This wound is handled with the greatest facility by transposing the lateral residuum of the tongue into contact with the mucous membrane of the ipsolateral cheek and effecting a direct closure (Fig. 1). The effect of this maneuver is most satisfactory in that the wound is immediately closed. Indeed, wounds resulting from extensive resections of these organs in the oral cavity may be closed with reasonable assurance of healing per primam. The residuum of the tongue has adequate function for eating and talking. This facility is explained by the fact that the tongue, being sutured to the cheek, which is a flexible and mobile part of the face, is not restricted in its movements. The esthetic deformity, which is always present in this type of problem, varies from slight to moderate depending upon the amount of tissue excised and whether an implant is inserted into the mandibular arch. When the amount of tissue excised is extensive and there is some doubt about the adequacy of tissue for closure or when the tissue bed has been heavily irradiated, an immediate bone grafting or inert implant is not warranted. When there is adequate residual tissue in the oral cavity to cover an immediate bone graft and the tissue bed is healthy,

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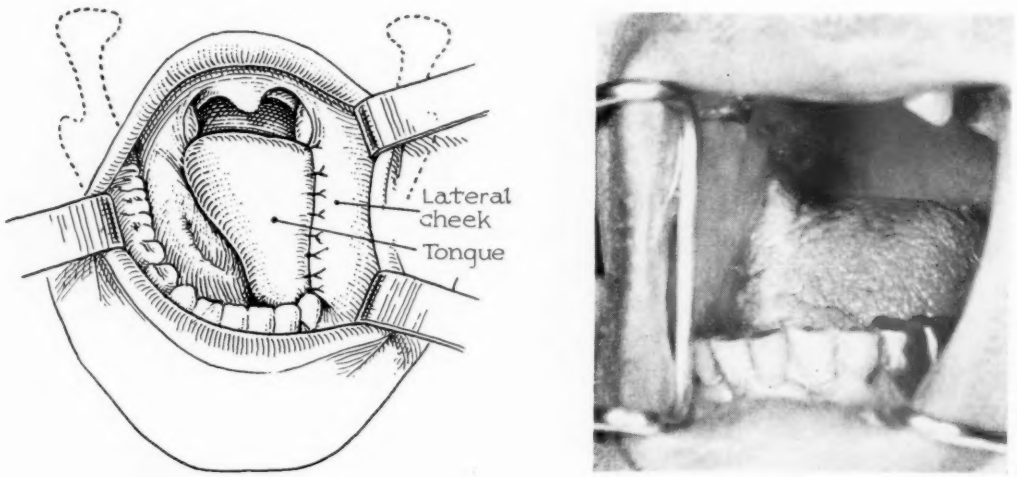
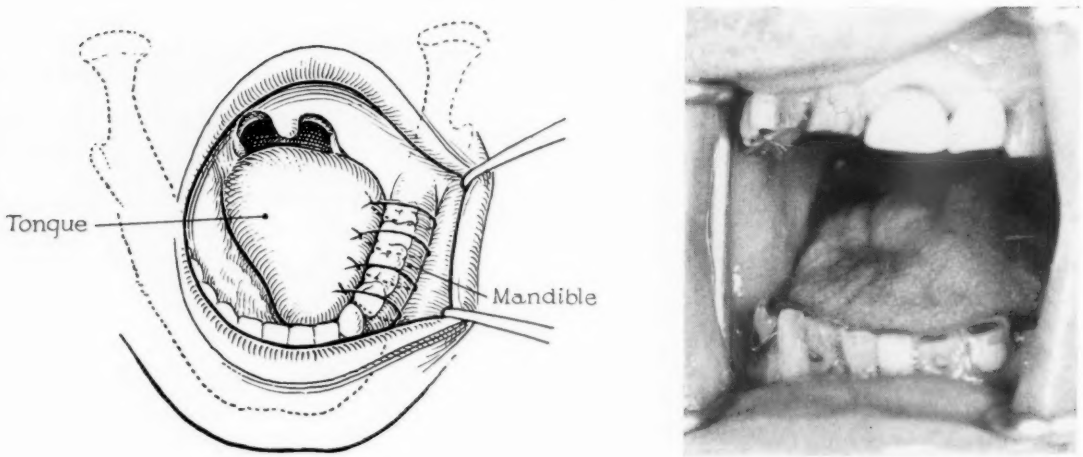
the outlook for immediate restoration is excellent. In this latter circumstance the tongue is used to cover and nourish the superior and medial aspects of the bone graft.

Tongue to Mandible.—Where there is a limited cancer involving the floor of the mouth or the undersurface of the tongue and the mandible is not involved, these soft tissue areas are excised in conjunction with the lateral neck



A. *B.*

Fig. 1.—*A* and *B*, Tongue to cheek.



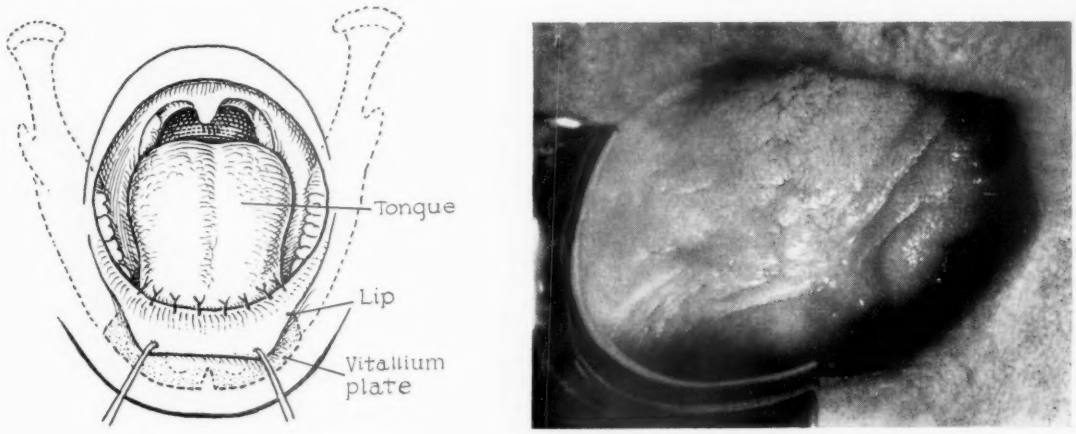
A. *B.*

Fig. 2.—*A*, Tongue to mandible. *B*, Residuum right tongue approximated to mandible.

and suprahyoid contents by the "pull through" technique. The resultant intraoral wound is in approximation with the inner aspect of the mandible, the tongue, and neck. This wound is most expeditiously handled by transposing the lateral residual portion of the tongue into the concave aspect of the intact mandibular arch. It is secured in this position by large circumferential sutures about the tongue and mandible causing an approximation of the raw

edge of the cut tongue to the mandible (Fig. 2). This eliminates the dead space in the oral cavity and suprahyoid area and has the advantage of permitting the mandibular arch to remain intact. The functional qualities of the tongue are moderately restricted following this method of closure, primarily because the residuum of the tongue is attached to the mandibular arch which is a fixed structure that encumbers the intrinsic mobility of the tongue. This disadvantage, however, can be corrected at a later date by the implantation of an epithelial skin graft into the glossoalveolar sulcus. The technique, on the other hand, has the advantage of a normally functioning mandible with practically no impairment to esthetics.

Tongue to Lower Lip.—Extensive cancers of the anterior floor of the mouth, tongue, mandible, and neck require radical resection of all of these associated structures. Extensive wounds in the midanterior portion of the lower third of the face, if not repaired adequately, cause great interference with function and esthetics. Inadequate repair of the patient who has had the floor of



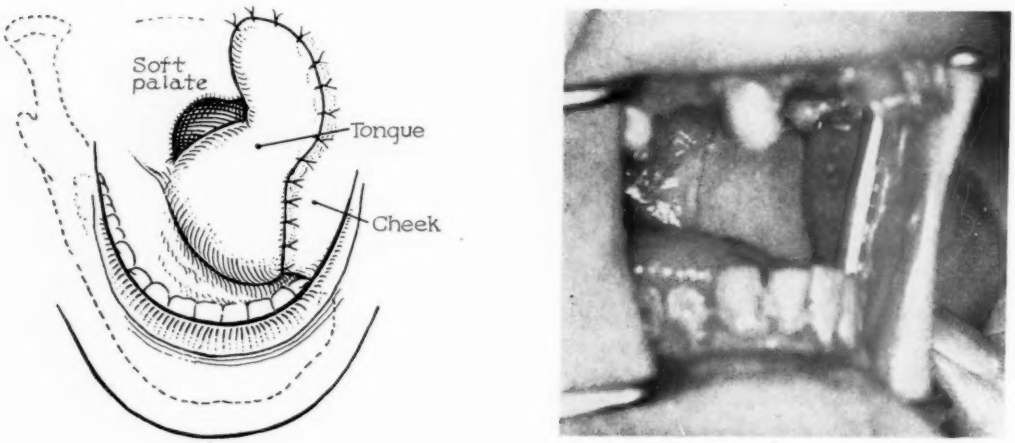
A. *B.*

Fig. 3.—*A* and *B*, Tongue to lower lip.

the mouth, mentum of the mandible, and a portion of the tongue and neck removed led to the use of the term "Andy Gump deformity." A satisfactory method of handling these wounds consists in the transposition of the anterior portion of the residuum of the tongue to the inferior aspect of the mucous membrane of the lower lip. This accomplishes a covering for the wound in the region of the floor of the mouth and the implant which has been inserted beneath the tongue for the reconstitution of the mandibular arch. (Fig. 3). The implant establishes stability to the mandibular arch and is often best accomplished by using an insert metallic prosthesis, such as Vitallium, stainless steel plate, or Steinman pin. Although bone grafts had been used in this instance, the difficulties of retaining them present a real hazard to their success. One should pay particular attention to the length of the superior neck flap so that it may be used to assist in obliterating the submental dead space. The results of the repair are satisfactory. The functional activity of the mandible is moderately

impaired. The esthetic result is good. An epithelial inlay skin graft may be carried out 3 to 6 months postoperatively to enhance the mobility of the tongue and create a deeper glossolabial sulcus.

Tongue to Soft Palate.—Extensive cancers involving the soft palate, lateral oropharynx, pharynx, tonsil, and ascending ramus of the mandible may be handled by resection of these associated structures. The magnitude of the intraoral wound precludes any attempt at reconstruction, and one is satisfied to attain healing per primam. These problems are divided into two classes, namely those where the resection of soft tissues can be carried out without interfering with the ascending mandible and those including the mandible. The latter situation is applied in the more extensive cases and may also include the pterygoid plate and muscles, soft palate, tonsil, partial pharynx, and lateral neck contents.



A. *B.*

Fig. 4.—*A* and *B*, Tongue to palate.

In the first instance the wound is best handled by transposing the lateral and posterior aspects of the tongue superiorly to the lateral aspect of the soft palate and pharynx. It may be necessary to make a small fillet of the tongue to accomplish this. In the second case, repair by this method is impossible, as the diameter of the oral pharynx would be so small it would prevent swallowing. Here a large fillet of the ipsilateral half of the tongue is rotated into the palate, pterygoid area, and pharynx as a flap to cover the wound (Fig. 4). Function of the oral cavity is impaired to a moderately serious degree in most of the advanced problems. Esthetics are only mildly disturbed. Function may be improved in either class by an epithelial inlay graft between the tongue and palate after a 3 to 6 month interval.

Tongue to Lateral Pharynx.—The treatment of cancers of the lateral pharynx and base of the tongue includes the radical excision of these organs in continuity with the lateral neck. The lateral pharyngeal wound is immediately repaired by the transposition of the posterior and inferior cut border of the tongue to the remaining mucous membrane in the oro- and hypopharynx. A vertical fillet of the base of the tongue may improve the contour and increase

the diameter of the hypopharynx. If half of the tongue remains in the wound, the functional capacity of the oral cavity and pharynx is usually good, although there may be considerable difficulty in rehabilitating the act of swallowing if the larynx remains in situ. If the larynx has been removed with the specimen, swallowing is ordinarily no problem. Esthetics are not seriously involved. If the pharyngeal or hypopharyngeal defect is so extensive that the repair of the wound by transposition of the tongue would compromise either swallowing or respiration unduly, then a free inlay epithelial skin graft is applied over the prevertebral fascia or the creation of a pharyngostome should be considered.

Tongue to Anterior Pharynx (or Cervical Esophagus).—The treatment of extensive cancers of the base of the tongue, pharynx, epiglottis, larynx, and postericoid area is the radical extirpation of these sites in conjunction with neck dissection. The immediate repair of the more limited type of wound created by this excisional technique is carried out by either the vertical T type of mucous membrane repair of the pharynx and base of the tongue or the horizontal crescent shape repair of the hypopharyngeal mucosa to the base of the tongue. If the wound is extensive, however, there may be a paucity of mucous membrane.

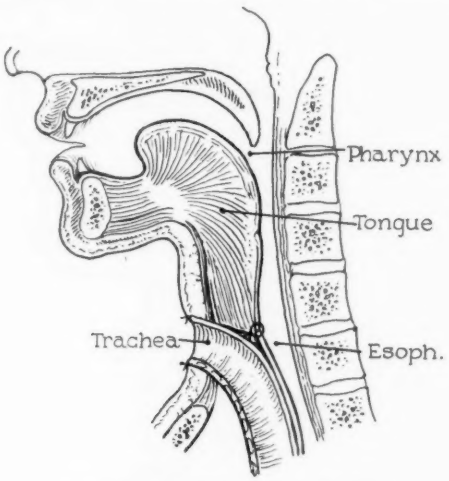


Fig. 5.—Tongue to esophagus.

Under these circumstances one can usually attain primary closure by transposing the base of the tongue inferiorly to the hypopharynx and, upon certain occasions, to the upper portion of the cervical esophagus (Fig. 5). This latter maneuver is aided by marked flexion of the head and neck during the repair and holding the neck in the flexed position for a period of 10 days postoperatively. This transposition is more flexible in the absence of the hyoid bone which receives considerable suspension from the stylohyoid and mandibular ligaments and muscles. The functional capacity of the oral cavity and pharynx is usually normal.

Tongue to Pharyngostome.—Extensive cancers involving a portion of the tongue and the major portion of the pharynx may be treated by partial glossectomy, pharyngectomy, and neck dissection. In some instances it is also necessary to resect the lower lip and mandible. If the larynx is compromised it is

electively removed. In these unusual circumstances, it is often impossible to carry out an immediate complete reconstruction of the wound. In such instances the residuum of the tongue is a valuable organ to be used as a dressing for the anterior inferior third of the face on the lateral neck, when there is inadequate skin and mucous membrane. It is anticipated that the tongue be released

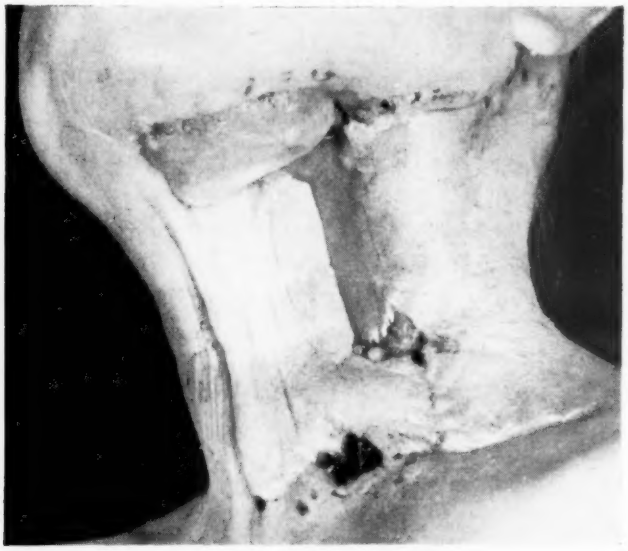


Fig. 6.—Tongue to pharyngostome.

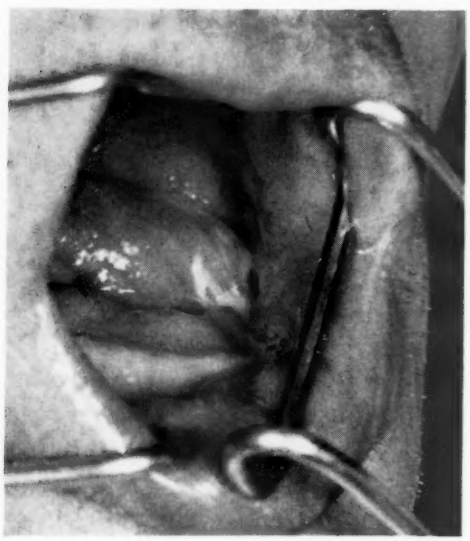


Fig. 7.—Left half of tongue transposed into palate and nasopharynx.

in a matter of 2 to 4 weeks from its primary manipulation for expediency and repositioned into the oral cavity with the subsequent substitution of adequate transposed pedicle flaps for the creation of lip, lateral neck, and closure of pharyngostome cavities. Both function and esthetics are seriously altered during the preliminary stage of wound treatment. However, these are improved

after adequate tissue replacement is brought into the wound in the second or third stage. The prognosis is obviously grave in cancers of this magnitude. (Fig. 6.)

Tongue for Coverage of the Carotid Vessels.—Recurrent cancer following intensive irradiation involving the area of the soft palate, pterygoid structures, lateral pharynx, tonsil, and mandible is best handled by radical resection of these sites in conjunction with neck dissection. The resection of these structures causes an exposure of the common and internal carotid arteries in connection with the pharyngeal and nasopharyngeal spaces. In some instances there may not be adequate supply of epithelial covering remaining in the neck to cover the carotid bulb adequately. Under these circumstances the tongue may be filleted and transposed over the artery to protect it from the hazards of postradical surgery and postirradiation slough in the presence of saliva and secondary infection. When the recurrent tumor is primarily in the region of the soft palate, tonsil, and pterygoid area, the problem of carotid artery coverage is a more difficult one. The internal carotid artery may be exposed from the bulb to the base of the skull. There is never enough remaining mucous membrane in the wound to effect a closure per primam. Again, in the postirradiated, postradical surgery problem the artery may be covered by transposing a fillet of the lateral border of the tongue superiorly over the artery, thus providing a protective covering against secondary infection and saliva and affording adequate nourishment to a vessel that might otherwise carry a high potential for eventual ulceration and spontaneous rupture as the postirradiated wound heals by secondary intention. This technique carries a moderate impairment to function of the oral cavity but no essential esthetic alteration. (Fig. 7.)

SUMMARY AND CONCLUSIONS

1. Eight varieties of circumstances resulting from radical surgery in the head and neck present situations that are best handled by mobilization, transposition, or fillet of the tongue.

2. These situations include approximation of the tongue to the cheek, mandible, lower lip, soft palate, lateral pharynx, and anterior pharynx (or esophagus) and the use of the tongue in pharyngostomes and for carotid artery coverage.

3. The advantages of these various techniques include immediate closure of the wound, limited interferences with oral function and esthetics, and reduced morbidity.

THE THEORY OF PROTEASE ACTIVATION AND ITS ROLE IN THE REJECTION OF HOMOTRANSPLANTS

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THE rejection of homotransplants has been considered to be due to immune response of the host. Since the destruction of tissue is involved, it may be appropriate to postulate a hypersensitive reaction. The main concepts of hypersensitivity deal with (1) the relatively important role of circulating antibodies, (2) the cellular sites of reaction of the antigen-antibody combination, and (3) the activation of proteolytic enzymes which directly or indirectly produces injury of sensitized cells. To date, no one has demonstrated circulating antibodies or passively transferred the reaction with either serum or cellular elements.

The protease activation theory of hypersensitivity may offer a new concept in explaining the possible chain of events leading to the rejection of a homotransplant. Protease activation is thought to be the result of an initial antigen-antibody reaction which stimulates or "activates proteolytic enzymes and thereby causes injury or death of cells."[1, 3, 7]

In order to test the feasibility of this theory a study of protease activity was done in skin grafted mice. Burdon[2] has shown in experiments with hypersensitivity that there is a rise in circulating serum protease which was followed by an antiprotease after sensitization with certain proteins. Burdon's film strip method was used to demonstrate antitryptic activity in our experiments.

EXPERIMENTS

The antitryptic titer was determined on pooled sera obtained at various intervals from 310 6-week-old CFW mice. Shaved full-thickness autografts and homografts weighing 160 mg. folded with the raw surface out were implanted subcutaneously.

The following groups of animals were studied. (1) Six titrations on pooled sera from 40 normal mice were done for normal values. (2) Fifty subcutaneously autografted mice served as a control series. (3) Sixty mice were homotransplanted subcutaneously. (4) Twenty mice received 300 r. whole-body irradiation. (5) Sixty mice received 300 r. irradiation and were subcutaneously homografted 24 hours later. (6) Sixty mice were injected subcutaneously with 0.25 c.c. of a 10 per cent suspension[6] homologous skin extract and homografted with a subcutaneous implant 7 days later. (7) Twenty mice were injected with 0.25 c.c. of a 10 per cent homologous skin extract.

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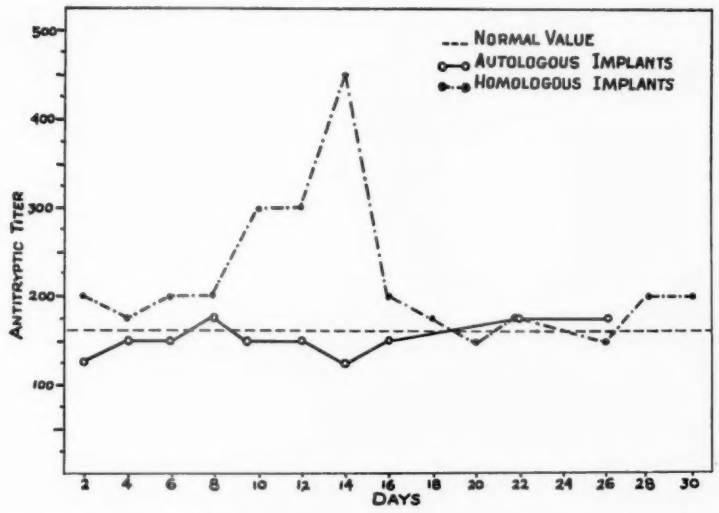

Fig. 1.—Antitryptic titers of mice receiving autologous and homologous implanted skin.

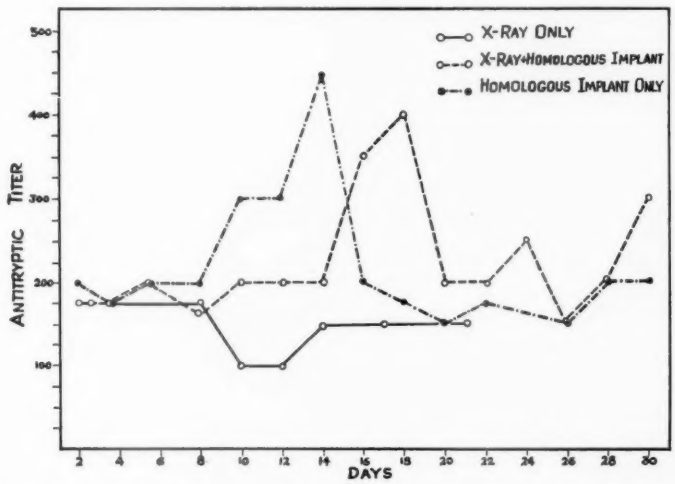

Fig. 2.—Antitryptic titers of mice receiving irradiation and homologous skin implants.

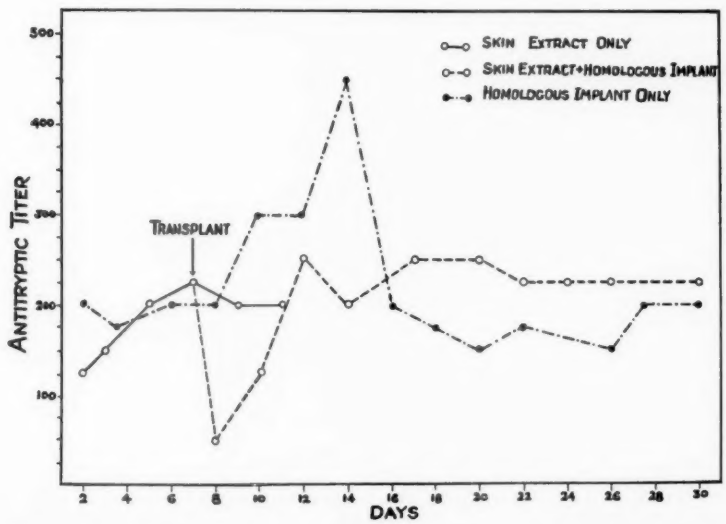

Fig. 3.—Antitryptic titers of mice receiving skin extract and homologous skin implants.

RESULTS

The antitryptic titer for normal CFW mice was between 150 and 175. Autogenous skin implants showed a response well within the normal values (125 to 162). However, in mice implanted with homografts the antitryptic index rose sharply on the eighth day, reached a peak of 450 on the fourteenth day and thereafter returned to normal (Fig. 1).

Irradiation alone produced a depressed titer on the eighth to tenth day which returned to a normal level on the fourteenth day.

Irradiation with a subcutaneous homograft showed a progressive increase of the antitryptic titer during the first 2 weeks with a rapid elevation to 400 by the eighteenth day which gradually returned to normal (Fig. 2).

Animals injected with skin extract showed a slow but sustained rise in the antitryptic titer to 200. When the homografted implant was added, the titer dropped precipitously in 24 hours, rose to 250 by the twelfth day, and then assumed a nearly normal level (Fig. 3).

DISCUSSION

The antitryptic titer showed no activity after autotransplantation but showed marked elevation after homografting. Previous experiments have shown that surface homografts show rejection and necrosis on the sixth to eighth day and the sloughing of the graft is completed on about the fourteenth to eighteenth day.[5, 8] The observed rise in antitryptic titer closely paralleled the necrosis and rejection of the implanted homograft (Table I). Autologous implants on microscopic study were viable for the 30-day observation period. However, homologous implants showed beginning necrosis at 8 days with no persisting viable epithelium after 16 days. Total body irradiation and a homologous implant delayed the onset of necrosis for an additional 4 to 6 days. In the irradiated group there was no persisting viable epithelium of the homologous implant after 18 to 20 days. Skin extract and a homologous implant again delayed the onset of necrosis to 11 to 12 days. Viable epithelium in this group of animals persisted the same length of time as in the irradiated group. Since the protease activation theory postulates that the trypsin inhibitor is produced in response to the elaboration of proteases resulting from an antigen-antibody reaction, then the antitryptic titer may be interpreted as an expression of antibody levels.

TABLE I. FATE OF IMPLANTED SUBCUTANEOUS GRAFTS

	BEGINNING NECROSIS AFTER GRAFTING (DAYS)	PERSISTING VIABLE EPITHELIUM (DAYS)
Autologous graft	None	Viable
Homologous graft	8	16 but none thereafter
Irradiation and homologous graft	12-14	18-20 but none thereafter
Skin extract and homologous graft	11-12	18-20 but none thereafter

The depression of the antitryptic titer following whole-body irradiation may be explained by injury to the reticuloendothelial system and its subsequent depression of antibody production.[6] Substantiating evidence is given

to this theory by the results observed on serum proteins in mice who have received total body irradiation. Gamma globulin determinations made from 18 hours to 2 weeks after irradiation showed a consistent depression with a gradual return to normal.[9]

Whole-body irradiation delayed the onset of necrosis of the homografted implant (12 to 14 days). The antitryptic titer correlates well with the microscopic findings of the graft.

The over-all effect of the injection of skin extract alone as well as with a homograft implant showed a slight but persistent rise in the titer (200). The subcutaneous route of the skin extract alone seemed to exert a slow but gradual source of stimulating antigen as compared to an implanted homograft. The implantation of a homograft 7 days after subcutaneous skin extract appeared to provide an additional source of antigen. The summating effect of this skin graft antigen caused a sharp decrease in the antibody response as reflected in the depression of the antitryptic titer. During the next 4 days the antitryptic response returned to an elevated but level range. The effect of skin extract and homografted implant differed in that the beginning necrosis (11 to 12 days) was not reflected in a sharp rise in the antitryptic titer as compared with a homografted implant alone or with irradiation and implanted homograft. The injection of skin extract subcutaneously seemed to exert a different response from that of a subcutaneous implanted homograft.

SUMMARY

The initial sensitization of the host by a homograft is thought to activate proteolytic enzymes which cause the death of the transplant. The theory of protease activation may offer an explanation for the host's rejection of a homograft. The antitryptic titers in these experiments closely parallel the necrosis and death of the homografts. Irradiation or the injection of skin extracts depressed the antitryptic titer and prolonged the life of the homograft.

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RESULTS OF PORTACAVAL SHUNTS IN THE TREATMENT OF PORTAL HYPERTENSION

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THIS communication will review an experience of 6 years in the treatment of portal hypertension by portacaval shunts. It should not be necessary to emphasize the seriousness of hemorrhage from esophageal varices and therefore the necessity of avoiding this complication of portal venous system block. The mortality rate in patients who bleed from esophageal varices ranges from 50 to 70 per cent.[22, 25, 31] An entirely satisfactory method for dealing with patients while they are bleeding has not yet been achieved. Prevention of hemorrhage is the best hope for these patients. A portacaval shunt performed at a time when the patient is in the best physical condition is the most satisfactory surgical procedure available. Any discussion on this subject, however, must first pay tribute to the work of Blakemore[6] and Whipple[32] who introduced this technique of portal venous bed decompression in the treatment of patients who have bled from esophageal varices. Since the first report of Blakemore and Lord,[6] others have contributed to knowledge in this field and added testimony to the soundness of the concept of portal-systemic venous shunts as applied to the treatment of portal hypertension and its complications.[4, 5, 8, 13, 15, 16, 19, 30]

The most significant information which was obtained in this study has been the over-all results which have followed portacaval shunts, in terms of the prevention of bleeding from esophageal varices in patients surviving operation. The immediate mortality rate of portacaval shunts and the factors bearing on its improvement over the years is another interest which this review of patients will present. This latter matter has a very important place in the consideration of whether or not these operations are to be generally recommended. Experience in the management of the patients in this series has also brought some light to bear on the efficiency of portacaval shunts versus splenorenal shunts. These, and a number of other items bearing upon the whole problem of portal hypertension, particularly when the latter is caused by liver disease, will be discussed.

CLINICAL MATERIAL

This study group is made up of 40 consecutive patients upon whom 41 portacaval shunts have been performed by one of us (C. S. W.), during a 7-year period, the years July, 1949, through July, 1955, inclusive.* Every

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*These operations were performed at the New England Center Hospital, Boston, Mass., the Albany Hospital and the Albany Veterans Hospital, Albany, New York.

patient has been followed and information is available up to July, 1955. In Table I a summary has been made of the general points of interest which pertain to patients who were subjected to portacaval shunts. Some of these data will be referred to as they bear on the diagnosis, indications for operation, and selection of patients for surgery.

TABLE I. GENERAL DATA ON FORTY PATIENTS SUBJECTED TO PORTACAVAL SHUNTS

AGE	
Average age, 42 years	
Range, 10 years to 65 years	
Eighty per cent over 40 years	
SEX	
Males 42%; females 58%	
ETIOLOGY OF PORTAL HYPERTENSION	
Intrahepatic obstruction	
Alcoholic cirrhosis	50%
Posthepatitis cirrhosis	38%
Other liver disease	4%
Extrahepatic obstruction	8%
ESOPHAGEAL VARICES	
Positive evidence of varices	73%
Equivocal evidence of varices	5%
No evidence of varices	20%
Incomplete or no studies for varices (one patient)	2%
HEMORRHAGES	
One or more hemorrhage	92%
No hemorrhage	8%
HYPERSPLENISM	
Splenomegaly with cytopenias	50%
ASCITES	
Present in 25%	

DIAGNOSIS AND INDICATIONS FOR SHUNT SURGERY

The diagnosis of portal hypertension is not difficult to make in all but a few patients. All patients with cirrhosis of the liver have some degree of increased portal venous stasis, but the magnitude of hypertension varies depending upon the completeness of the venous obstruction and the facility of collateral circulation.[13, 15] It is an established fact that extensive collateral venous channels other than by the veins of the esophagus exist in patients with portal hypertension. These veins of which the retroperitoneal groups are the most numerous do not tend to rupture and bleed, however. That the varices at the gastroesophageal junction bleed is probably the result of a number of factors. They are larger than other collateral vessels and are exposed to the trauma of the mechanical and chemical digestion of food.[2] Erosion of the mucosa at the esophagogastric junction may play a large part in initiating hemorrhage. Esophageal varices should be looked for in all patients with cirrhosis of the liver since it is in patients with varices that massive hemorrhage occurs. Unfortunately, the diagnosis of varices is not made frequently enough before a hemorrhage jeopardizes life. Two other clinical entities

should bring up the question of portal hypertension and esophageal varices. The first presents in all cases of massive bleeding from the upper gastrointestinal tract. Some patients do not show overt evidence of liver disease and resemble patients bleeding from peptic ulcer. Secondly, whenever splenomegaly is encountered in a patient and especially when leukopenia and anemia are found with it, the possibility of congestive splenomegaly as the result of portal venous bed block should be entertained, and further studies of liver function, the esophagus, and portal venous systems should be made.

In the group of patients of this study all but 3, or 37, had disease of the liver or intrahepatic portal venous obstruction as determined by clinical tests before operation and liver biopsy at the time of surgery. Twenty-one, or half, of the patients had evidence of hypersplenism manifest variously by anemia, leukopenia, and thrombocytopenia singly or in combination. Ascites was present in 10 patients but was lessened or made to disappear as liver function was improved under medical treatment, or by intravenous repair of the serum protein by blood or plasma transfusions. Although portal hypertension is not the cause of ascites in patients with liver disease, it is a contributing factor to its magnitude, especially when the values for serum albumin are low.[9] This effect of portal hypertension was observed in several patients who exhibited ascites after each hemorrhage but who lost their ascites after blood replacement.

The fully accepted indications for surgery in patients with portal hypertension are a history of a previous hemorrhage and evidence that bleeding has occurred from esophageal varices. Whether indications should be broadened to include patients who have not bled but who have varices is a matter of debate and this question will be discussed later. Indications for portacaval shunts may exist under certain circumstances in which varices have not been demonstrated and in some cases in which hemorrhage has never occurred. It is a fact that the presence of esophageal varices cannot be demonstrated in some patients who have bled. Esophagoscopic examination may reveal varices when the esophagogram is equivocal, but on the other hand, the esophagoscopist may not see varices when the x-ray reveals them. If a hemorrhage can be stopped by using gastroesophageal balloon tamponade there is good evidence that hemorrhage is of varices origin especially if the patient has cirrhosis of the liver and if no other lesion of the gastrointestinal tract can be demonstrated. Each patient must be evaluated individually as to the probable cause of hemorrhage and it is wrong to exclude categorically from consideration for a portacaval shunt a patient who has had a massive hemorrhage and who has all other criteria for the diagnosis of portal hypertension only because varices in the esophagus cannot be visualized by usual diagnostic methods. The development of the techniques for portal venography has contributed substantially in the diagnosis of questionable cases.

An indication for portacaval shunts also exists in patients in whom a splenectomy is done for the alleviation of hypersplenism even though no varices exist or the patient has not bled. This applies only to the group of patients

with congestive splenomegaly with portal hypertension proved at operation. Advantage should be taken of the large splenic vein for anastomosis and prevention of later hemorrhage at the time of splenectomy.

In these 40 patients the presence of esophageal varices was proved clinically in 29 (73 per cent) of the patients; in 2 patients the diagnosis of varices was equivocal. Evidence of portal hypertension was proved in all by a combination of clinical studies and actual measurements of portal system pressure at operation. Three patients subjected to portacaval shunts in this series had never bled and in each of these patients a splenorenal shunt was done at the time of a splenectomy performed for the relief of severe hypersplenism. Esophageal varices were not demonstrated in these 3 patients. All 3 had liver disease and portal hypertension proved at operation by pressure readings and liver biopsy. Of the 37 patients in whom hemorrhage had occurred only 6 had had but one previous bleeding episode.

SELECTION OF PATIENTS FOR PORTACAVAL SHUNTS AND THE PREPARATION FOR SURGERY

It goes without saying that patients who have survived a severe hemorrhage of portal hypertension origin should be protected against another such episode if the risk of the elective operative procedure is not too great. It is also a fact that more than a majority of the survivors of hemorrhage are candidates for portacaval shunts since their liver function has to be fairly good to survive a hemorrhage. Certain standards based on liver function studies have proved to be of value in selecting patients who can be expected to weather a major surgical procedure.[3, 10, 16] That the value for serum albumin should be 3 Gm. per cent or better is a good rule and in general can be applied. Values for the retention of the dye, Bromsulfalein, should be below 30 per cent. Patients with marked jaundice should rarely, if ever, be subjected to portacaval shunts since their problem is severe intrahepatic disease.

Any patients with persistent values for serum bilirubin of above 3 mg. per cent present evidence of severe liver disease. If the prothrombin time remains prolonged (5 seconds above normal standard) despite adequate vitamin K therapy, the patient is, again, a poor risk. Ascites that does not disappear or lessen is a contraindication to shunt procedures. All these statements presume that an adequate trial of good medical therapy has been carried out with insufficient improvement.

Almost as important as the actual values obtained in a study of the liver function in patients with cirrhosis of the liver is the observation of improvement in these values after relatively short-term treatment. Patients with adequate functional reserves in the liver will show improvement in from 10 days to 3 weeks. The Bromsulfalein retention value falls as does the serum bilirubin. The prothrombin time also improves. These three functional tests show rapid improvement in most patients who will prove to be acceptable candidates for shunts. An increase in the value for serum albumin comes more slowly; it can be raised temporarily by the administration of plasma or human serum albumin. An indication that the liver can make albumin on its own

must be evidenced either by a spontaneous increase of the value for serum albumin when none has been administered or by maintenance of an adequate level for some period after supplementary treatment.

Bed rest is imperative during preoperative treatment especially in patients who have recently had a hemorrhage. If ascites is present it should be relieved; patients feel better and eat better after paracentesis. A general diet or one high in carbohydrate is most palatable and acceptable to these patients. While it is desirable to have a good protein intake in order to serve the needs of repair of the serum albumin it is difficult for these patients to consume a very high protein diet. It also is undesirable to feed more protein than the liver can handle since the syndrome of meat intoxication may be brought on.[20, 21, 24] Blood ammonia nitrogen values should be taken from time to time to check this feature of dietary management. A diet low in sodium (1 or 2 Gm.) should be used if ascites is marked but if this diet is unpalatable it should be liberalized. Blood transfusions are usually necessary in the majority of patients, many of whom are anemic from a recent hemorrhage or from hypersplenism. Transfusions must be given with caution, and perhaps the hematocrit or hemoglobin should never be brought to what might be considered in normal people to be a desirable level. In our experience, massive transfusions of blood seem to precipitate hemorrhage from varices. It may be that the portal venous system is expanded by blood transfusion and that portal pressure is raised. Whatever the explanation may be for transfusions causing hemorrhage, it is safer to give small transfusions of 250 c.c. of blood per day as the normal blood volume is approached. Values for hemoglobin of 11 to 12 Gm. per cent and hematocrit values of from 37 to 40 per cent are adequate for operation. If the serum albumin is low and the hemoglobin is adequate, plasma is valuable in reaching an optimum physiologic state faster than can be accomplished by feeding alone. The prolonged use of serum albumin or plasma for the treatment of ascites is useless, however, since ascites will not disappear unless the liver function improves.[9, 14] Vitamin K supplementation is usually necessary and vitamins B and C administrations are used empirically. The administration of 1,000 c.c. of 10 per cent dextrose intravenously daily 7 to 10 days before operation is helpful. An intravenous pyelogram should be performed and an estimation of the excretion of phenolsulfonephthalein made to determine kidney function if hypotensive anesthesia is to be used. In addition, if a splenorenal shunt is contemplated, the presence of a functioning right kidney is important information.

Ninety per cent of the patients in this group were operated upon with values for serum bilirubin of 2 mg. per cent or less. In 10 per cent of the patients the values were between 3 and 4 mg. per cent. Likewise, 90 per cent of the patients had 3 Gm. per cent or more of albumin just prior to surgery. Ninety-two per cent had adequate prothrombin levels (15 to 20 seconds with the control at 15 seconds). Thirty-three per cent of the 40 patients in this group had values for Bromsulfalein ranging from 25 to 35 per cent. By and large, then, chemical evidence of adequate liver function was present

in 90 per cent of these patients in this group (counting 3 patients with extrahepatic portal obstruction and normal liver function). Only one quarter of the group had such good liver function when first seen and the final preoperative values obtained followed a period of 7 to 64 days of preparation.

OPERATIVE PROCEDURES AND RESULTS OF SURGERY

Forty-one portacaval shunts were performed on 40 patients. One patient had first a splenorenal shunt and later a portacaval shunt for recurrence of bleeding. In Table II certain general data on the operative procedures are summarized. Six patients died in the hospital after operation from a few hours to 36 days, making the operative mortality rate 15 per cent over all. Four of these deaths occurred among the first 20 patients and only 2 among the last 20 patients, making the present postoperative mortality rate approximately 10 per cent. The causes of death immediately postoperatively were as follows: 2 patients died of hemorrhage at the operative site (these were among the earlier cases); one patient died of thrombosis in the entire portal system apparently arising at the operative site; 3 patients died of liver failure.

TABLE II. FORTY-ONE PORTACAVAL SHUNTS PERFORMED ON FORTY PATIENTS

SHUNTS	NUMBER	POSTOPERATIVE DEATHS	RECURRENT BLEEDING	LATE DEATHS	TOTAL SURVIVORS
Portacaval (end-to-side)	19	4	1	3	12
Portacaval (side-to-side)	4	0	0	1	3
Splenorenal	17	2	6	6	8
Mesenteric-renal	1	0	1	1	0
Total	41	6 (15%)	8 (20%)	11	23 (58%)

Twenty-three patients had a portacaval shunt performed, and of these 19 were end-to-side and 4 were side-to-side. Seventeen patients had splenorenal anastomoses, and in one patient a mesenteric radical was anastomosed to the renal vein for lack of either a portal or splenic vein.

The selection of one type of shunt over another is largely concerned with whether a splenorenal or portacaval anastomosis should be done. A general statement can be made that all patients with extrahepatic portal obstruction should probably have a splenorenal shunt planned for them since the portal vein is often obliterated or otherwise useless in this syndrome. Portovenographic findings may dislose a portal vein; if so, a choice of shunts is open in patients who do not have intrinsic liver disease.[7, 26, 27] It is also usually true that marked splenomegaly is the rule in this group, and hypersplenism is a common complication which may make splenectomy advisable and thus a splenorenal anastomosis preferable. In patients with intrahepatic portal block from cirrhosis of the liver the surgeon usually has a choice as to whether a splenorenal or portacaval shunt should be done. In the 17 patients in this series who had splenorenal shunts, 14 were done for cirrhosis of the liver and 3 for extrahepatic obstructions. It was believed at the time at which these patients were operated upon that the large spleen and hypersplenism demanded splenectomy and a splenorenal shunt in these 14 cirrhosis

patients. This indication for splenorenal shunt, however, has been largely abandoned. In the last 18 patients of the series, only 2 splenorenal shunts have been performed. Direct portacaval shunt is mechanically superior to the splenorenal anastomosis. It is larger and less likely to become angulated. It is also more easily performed and, finally, the results are better in terms of the prevention of bleeding. In one patient with extrahepatic obstruction, previously mentioned, the splenic vein was not available because splenectomy had been performed and a branch of the inferior mesenteric vein was used. Bleeding promptly recurred in this patient. Shunts made with mesenteric veins of lesser size than the splenic vein are probably useless. They thrombose almost immediately and it is only good fortune if patients do not bleed after such shunts. The operation of ligation of esophageal varices or esophagectomy is probably preferable to shunts smaller than portacaval or splenorenal anastomoses.

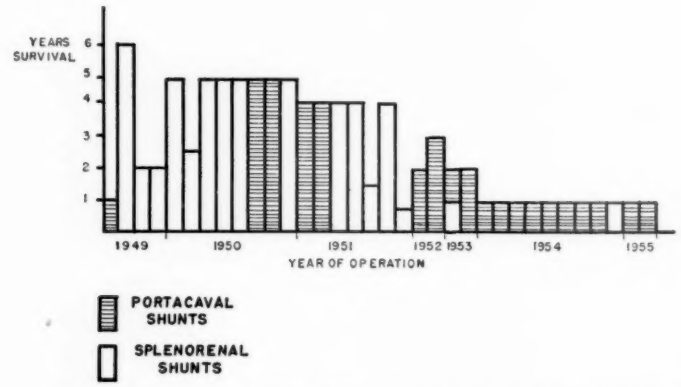

Fig. 1.—Survival time of 34 patients with portacaval shunts.

A follow-up study of this series shows that only one patient with a portacaval shunt bled after this operation. This patient did not die of hemorrhage. He was explored, however, and the shunt was found closed. This patient was a 14-year-old postsplenectomy bleeder and only a small portal vein was found. The incidence of postshunt bleeding for portacaval shunts is therefore 5 per cent in this series. Among 17 patients having splenorenal shunts, 6 have bled after operation, an incidence of 40 per cent. The high incidence of bleeding after splenorenal shunts can be attributed in part to the fact that 3 of these anastomoses were performed in children and were judged at operation to be far from satisfactory because of their small size. Nevertheless, the fact that splenorenal anastomoses thrombose more readily than portacaval shunts seems established.

The incidence of bleeding in all patients after shunting was 20 per cent, a figure higher than reported by other surgeons.[3, 8, 17] In addition to 6 postoperative deaths, 11 patients have died since their portacaval shunts. There are therefore 23 survivors out of the 40 patients (58 per cent) over a period of 1 to 6 years. Two postsplenorenal shunt patients who bled were subjected to esophagectomy and one to portacaval shunt at a later date. In Table III

the causes of death in patients who died one year or more after a shunt procedure are listed. There were 11 such patients. Hepatic failure heads the list and accounts for 6 deaths. Four of these patients had had portacaval shunts and 2 had had splenorenal shunts. Three deaths were due directly or indirectly to hemorrhage and all of these patients had had a splenorenal shunt. Two patients died of other ulcerative disease of the gastrointestinal tract. These data further support the view that splenorenal shunts are not as efficient as portacaval shunts. At the same time it must be admitted that portacaval shunt cannot be performed in all patients. It should also be pointed out that there are 6 patients in this series with splenorenal shunts and cirrhosis of the liver that have survived and are in good condition 3 years or more without recurrence of bleeding. In Fig. 1 the follow-up life span for each patient operated upon during each yearly period is recorded. Finally, among 34 patients surviving portacaval shunts only 3 died directly as the result of hemorrhage from esophageal varices and portal hypertension.

TABLE III. LATE CAUSES OF DEATH IN ELEVEN PATIENTS WITH PORTACAVAL SHUNTS

	TOTAL	SPLENORENAL	PORTACAVAL
Hepatic failure	6	2	4
Hepatic failure and hemorrhage	2	2	
Hemorrhage from varices	1	1	
Gastric ulcer hemorrhage	1	1	
Chronic ulcerative colitis	1	1	
Total	11	7	4

Six of the 11 patients who died at a late date after a shunt procedure were examined at post mortem and all were found to have a thrombosed splenorenal shunt even though hemorrhage was not the cause of death in each case.

DISCUSSION

Several advances have been made in the operative technique of portacaval shunts since 1945. These improvements in technique have been as important as those concerned with the preoperative and postoperative care of these patients. Operative exposure by using a combined abdominothoracic incision for either the right- or left-sided approach was the most important early advance. This was first described in the literature by Satinsky.[28] Preservation of the kidney by temporarily occluding its blood supply and the performance of an end-to-side splenic renal vein anastomosis followed by restoration of the kidney circulation was an important advance over the original splenorenal anastomosis which required nephrectomy. Linton[18] first published this technique using a left-sided abdominothoracic exposure. In portacaval anastomosis the original procedure described by Blakemore and Lord[6] required temporary occlusion of the vena cava above the renal veins while a nonsuture Blakemore cuff anastomosis was used to join the portal vein to the cava. This was cumbersome and dangerous. The use of a partially occluding caval clamp which allowed free flow of renal vein blood was then introduced into the technique.[29] The portacaval anastomosis could now be done almost at leisure

since the renal veins were not obstructed. This technique had been used in animal experiments years ago and was originated by Enderlen of Heidelberg.[11] Other smaller refinements of technique have also crept into the operation making a safer and faster procedure. Linton first used hypotension during portacaval shunt surgery employing Gillie's technique of continuous spinal anesthesia.[12] The advantages of hypotensive anesthesia have been enormous in two respects. The blood loss, and therefore blood replacement, has been markedly reduced because there is far less bleeding. The operative time has been reduced. Among the disadvantages of massive transfusions are the diminished coagulability of the blood at operation and the later complication of hepatitis.

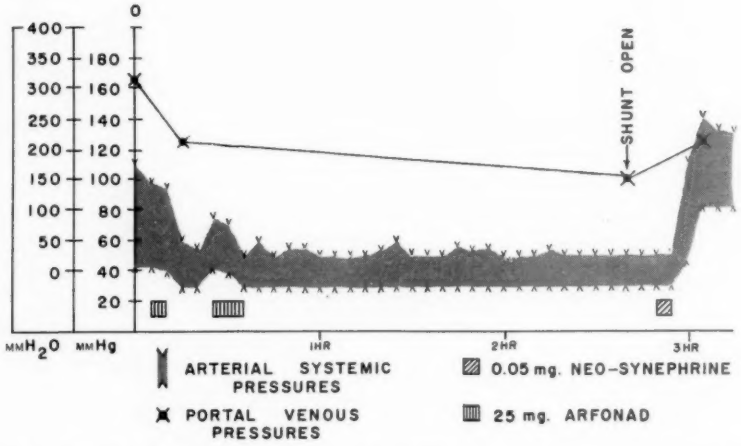

Fig. 2.—Effects of arterial hypotension on portal venous pressure. Case B 85248.

This technique of hypotensive anesthesia has been used in 8 of the patients in this series. Seven patients were anesthetized using a continuous spinal technique and one using Arfonad. There were no deaths in the group. Fig. 2 is a graphic representation of the changes in portal venous pressure during a portacaval shunt procedure using Arfonad. The continuous spinal technique is much to be preferred. It is more easily controlled and probably less dangerous. It can be seen that portal venous pressure decreases when systemic arterial pressure is low. This is a great boon to the surgeon in dissecting, making a more rapid operation. In Table IV the average blood loss and average operating time for 19 shunt procedures are shown (11 normotensive and 8 hypotensive). It is apparent that the blood replacement requirements are less using hypotension. The operating time is also shorter. It is now possible to perform most portacaval shunts in from 3½ to four hours using from 500 c.c. to 1,000 c.c. of blood. Hypotension had no deleterious effects on the

Table IV. Hypotensive Anesthetic in Portacaval Shunts

TYPE OF SHUNT	NUMBER	ANESTHETIC	OPERATIVE TIME (AVERAGE)	ESTIMATED BLOOD LOSS (AVERAGE)
Portacaval	11	Normotensive	5 hours, 15 min.	3,000 c.c.
Portacaval	8	Hypotensive	4 hours	850 c.c.

liver in these patients. It has been shown experimentally that liver blood flow is not reduced under hypotension when there is an adequate blood volume.[1]

Portal venography is another useful addition in the finite diagnosis of patients with portal hypertension.[7, 26, 27] It need not be applied routinely in all cases before operation by the method of percutaneous splenic injection of Urokon. The fact that cirrhosis of the liver is present is usually adequate for localization of the obstruction in the liver. It is especially useful when performed at operation to determine whether or not the portal vein is open and suitable for anastomosis in patients with apparent extrahepatic portal bed block in whom splenectomy has been previously performed. Not all patients with extrahepatic portal bed obstruction have an obliterated portal vein and venography has its best application in these cases.

The rather high rate of recurrence of bleeding in this series of patients (20 per cent) occurred almost exclusively among patients who had splenorenal shunts. It must be concluded that this shunt is more likely to thrombose than the portacaval type. There are many reasons why this should be so. The splenic vein has more branches and thus more technical difficulties are encountered in dissection. It is subject to more trauma during operation for the same reason. Kinking and angulation are also definite problems with the splenorenal shunt. A small splenic vein is another factor favoring thrombosis and closure. Nevertheless, the left-sided shunt has some usefulness in patients who need splenectomy because of marked hypersplenism. Decompression of a congested spleen by a direct portacaval shunt ultimately results in shrinking of the spleen and disappearance of hypersplenism but this effect may not be fast enough in patients with marked leukopenia and thrombocytopenia.

In considering the appalling mortality of hemorrhage from bleeding when it is the result of portal hypertension and from varices at the gastroesophageal junction it seems reasonable to us that portacaval shunts can serve a prophylactic role among patients who have not bled. At the Albany Hospital the mortality rate among patients with cirrhosis of the liver has been found to be 76 per cent at the time of the first hemorrhage. There is no second chance for many of these patients, therefore. The facts reported by others as well as the results in this series indicate that portacaval shunt protects against repetition of hemorrhage. It seems reasonable, therefore, that the prevention of hemorrhage by prophylactic portacaval shunt is a justifiable procedure in those patients with cirrhosis of the liver and esophageal varices who have never bled. Palmer, Brick, and Jahnke[23] have also expressed this opinion. This statement must be modified by stating that all patients must have the qualifications of adequate liver function and other criteria ordinarily applied in the selection of patients for shunts. In addition the separate circumstances of each patient have to be considered carefully. But, at any rate, it scarcely seems wise to wait for a hemorrhage to have occurred when advising a shunt procedure since bleeding is inevitable in these patients and occurs quite independently of the state of their general health or improvement in liver function. There is no convincing evidence that patients live for years with varices

and do not bleed. An occasional patient may be found with varices who has not bled, but no clinic or hospital has a sizeable group of patients with esophageal varices and portal hypertension under observation who have never bled. The occasional reports of patients who have had varices demonstrated by esophagogram with later studies indicating a spontaneous disappearance are no more reliable than the roentgenologic diagnosis of varices. It is a well-known fact that roentgenologists may differ in their opinion as to the presence and/or absence of varices in a patient at a given time and at different examinations.

Just how valuable portacaval shunts are for the type of patient who is currently being operated upon is a question recently raised.[8] There is no doubt that since it is among those individuals who survive a hemorrhage that patients for the shunt procedure are selected, liver function is better in this group than in those who succumbed during bleeding. It is not, therefore, a fair statement that patients who can be shunted after surviving a hemorrhage avoid a 50 to 80 per cent chance of dying from a hemorrhage at a 10 to 15 per cent risk of the shunt operation. But surgeons with experience in this field have not made any such claim. On the other hand, the fact remains, that at the Albany Hospital 66 per cent of 50 consecutive patients with portal hypertension died either directly or indirectly from their hemorrhage and 80 per cent of the same group were dead within one year. Similar experience has been reported by others.[22] It is true that hemorrhage may represent the termination of liver cirrhosis in some patients but it is not true that the very high mortality rate of hemorrhage from varices is largely due to the inclusion of patients at the end of their tether. Many of these patients are in sufficiently good condition a week before their hemorrhage that they could have been subjected to a portacaval shunt.

These, and other considerations, lead us again to the conclusion that, ideally, a shunt should be performed before hemorrhage occurs. If such a program were possible to carry out it might give us a truer picture of the value of portacaval shunts. Unfortunately hemorrhage may be the first indication of portal hypertension. Equally unfortunate is the current belief among physicians that hemorrhage is unlikely to occur so long as the patient is being maintained on a good medical regimen and showing improvement in liver function. A chance for a timely shunt is often lost by such reasoning.

SUMMARY

The data on 40 patients subjected to portacaval shunts over a period of 6 years have been presented.

The mortality rate for operation was 15 per cent among the entire 40 patients, but only 10 per cent for the 20 patients operated upon most recently.

The rate of recurrence of bleeding was 20 per cent; most of the patients with recurrent bleeding had splenorenal shunts.

The survival rate for all patients was 58 per cent.

Hypotensive spinal anesthesia used in patients undergoing portacaval shunts reduces blood loss and operating time and has no deleterious effect on the liver.

The suggestion is made that portacaval shunts should be performed on selected patients before they have ever bled if a greater benefit of this operation is to be applied to the problem of hemorrhage from esophageal varices in patients with portal hypertension.

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MANAGEMENT OF THE GASTRIC REMNANT AFTER SUBTOTAL GASTRECTOMY

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THE re-emergence of the older Billroth I gastrectomy in the face of the widespread popularity of the Polya-Billroth II is of considerable significance. Recent evidence suggests that those procedures which by-pass the duodenum predispose to certain nutritional deficiencies and may be a factor in postoperative anemia and failure to gain or maintain weight.

After an adequate gastrectomy, the Billroth I repair is not always easy, and undue tension may be placed on the gastroduodenal anastomosis. (Fig. 1.) A number of procedures have been proposed to obviate this eventuality and facilitate closure. These include the Kocher maneuver or mobilization of the duodenum, incision of the gastric omental attachments to the diaphragm, kidney, and liver, and incision of the esophagophrenic ligament with mobilization of the esophagus. It has even been suggested that the cystic duct be severed (with subsequent cholecystectomy) in order to gain more mobility for the common bile duct. Notwithstanding, there are instances in which mobilization fails to achieve sufficient length for comfortable anastomosis.

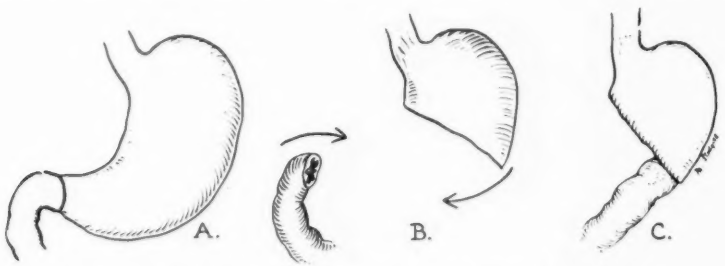

Fig. 1.—Subtotal gastric resection with Billroth I repair.

METHODS AND MATERIALS

In an effort to obviate the anatomic difficulties in gastroduodenostomy following high subtotal gastrectomy, several techniques of treating the small gastric pouch were explored. In the first method a simple incision was made in both the anterior and posterior wall of the pouch perpendicular to the line of resection after a high subtotal gastrectomy. (Fig. 2). Closure of the lesser curvature considerably lengthened the gastric remnant facilitating anastomosis with the duodenum. However, the resulting hour-glass configuration of the newly fashioned stomach was not considered ideal. In a second method a semicircular area of the gastric pouch on both the anterior and posterior surfaces

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was excised (Fig. 3). The cut margins of these surfaces were then united to each other with a continuous inverting mattress suture. This gave rise to a long, narrow viscus which provided adequate length for anastomosis to the duodenum.

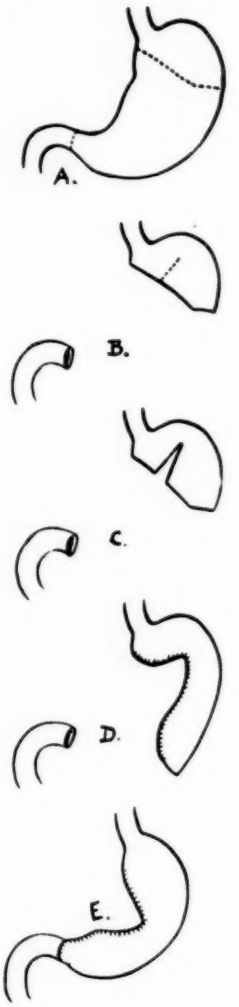

Fig. 2.

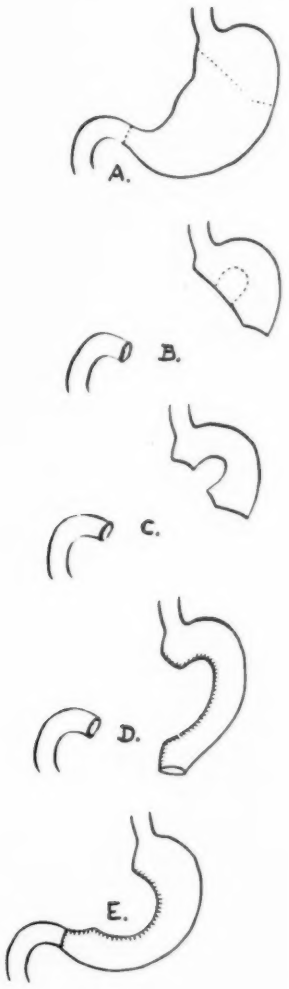

Fig. 3.

Fig. 2.—*A*, **Stomach and duodenum with lines of proposed resection.** *B*, **Subtotal resection completed.** *C*, **Incision in anterior and posterior walls of gastric pouch.** *D*, **The anterior and posterior walls are united along the previously placed incisions.** *E*, **The gastric pouch is anastomosed to the duodenum.**

Fig. 3.—*A*, **Stomach and duodenum with lines of proposed resection.** *B*, **Subtotal resection completed.** *C*, **Semicircular area excised from the anterior and posterior walls.** *D*, **The anterior and posterior walls are united along the previously placed incisions.** *E*, **The gastric pouch is anastomosed to the duodenum.**

In the third method (Fig. 4) a series of 5 mongrel dogs weighing 12 to 18 Kg. were used. A high subtotal gastrectomy was performed in which the greater curvature from the duodenum to a point halfway through the vasa breva and practically the entire lesser curvature were removed. The average dimensions of the excised specimen were 9 inches along the greater curvature

and 4½ inches along the lesser. Approximately 4 to 5 inches of the greater curvature was left; a narrow margin of the lesser curvature was left for the suture lines.

An incision was made on the anterior wall of the gastric remnant at the junction of the upper and middle thirds, and a similar incision was made on the posterior wall at the junction of the middle and lower thirds (Fig. 4, *C* and *D*). Closure of these staggered incisions using a modified Z-plasty technique (Fig. 4, *E* to *G*) provided a long, narrow pouch with as much length along the lesser curvature as was present on the greater curvature (Fig. 4, *H*).

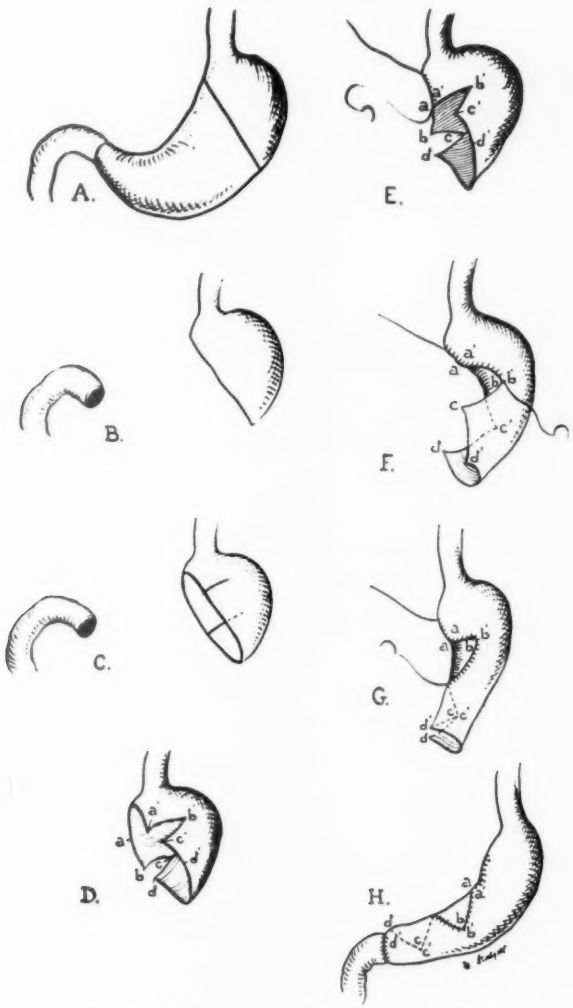

Fig. 4.—*A*, Stomach and duodenum showing lines of the proposed resection. *B*, Subtotal resection completed. *C* and *D*, Incisions made into the gastric remnant perpendicular to the line of resection. Anterior wall incision made at junction of upper and middle thirds; posterior wall incision at junction of middle and lower thirds. *E*, Closure of the resection is then begun at the upper or proximal corner and carried down one-third of the way to unite points a and a′. *F*, Closure of the staggered incisions are then begun by uniting point b (junction of middle and lower thirds on posterior wall) with point b′ at the apex of the incision on the anterior wall. *G*, Continuation of the suture line to unite point c with the apex of the posterior wall incision (point c′). *H*, Completed Z-plasty repair of gastric remnant and anastomosis to duodenum. Note lengthening of the lesser curvature side of the reconstructed stomach.

The animals were followed from 6 weeks to 6 months. Preoperative x-rays were compared with interval films taken postoperatively (Fig. 5). Adequate gastrectomy and functional gastric reservoir were demonstrated.

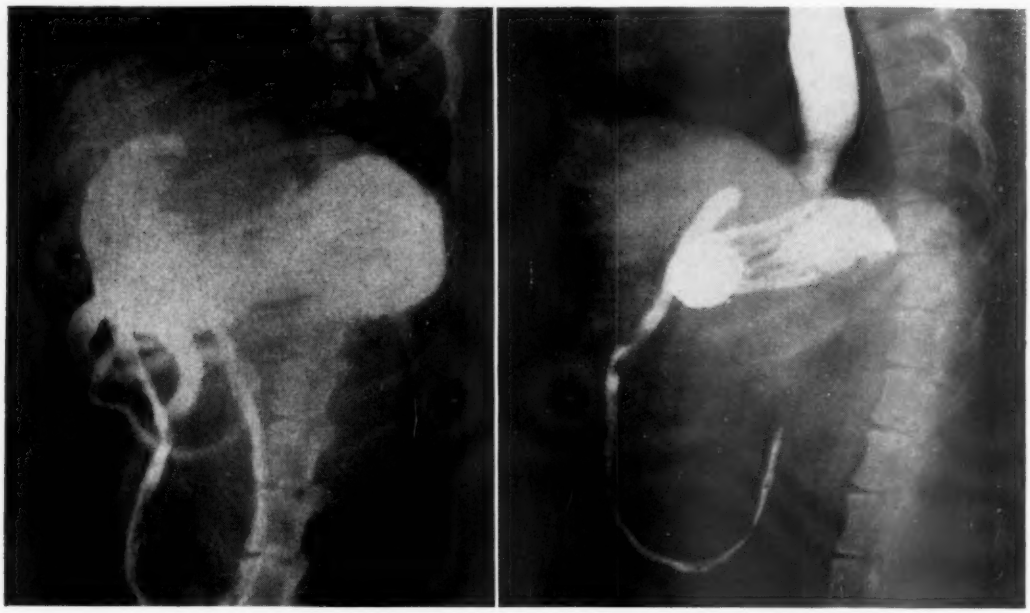

Fig. 5.—*Left*, Preoperative x-ray of stomach after barium meal, (Dog No. 24). *Right*, Same dog 6 weeks postoperatively. Barium demonstrates a small, but long, narrow gastric pouch. The attempt to completely distend this stomach is shown by forceful injection of barium into the esophagus.

DISCUSSION

Closure of the gastroduodenal anastomosis does not present the same problem in the dog as in man, because of the mobility of the duodenum in the former. Nevertheless, this experimental animal serves adequately to demonstrate the principle that the lesser curvature aspect may be readily elongated by the simple expedient herein outlined. It is hoped that with this introduced concept one need not resort to performing less than an adequate gastrectomy in order to allow gastroduodenal closure.

SUMMARY

A method for repair of the gastric remnant with gastroduodenostomy following high subtotal gastrectomy is described together with observation on the use of this technique in the experimental animal.

THE CHOLEDOCHODUODENAL JUNCTION IN THE CAT

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REVIVAL of interest in the sphincter of Oddi, following the discovery of a new intravenous method of visualizing the human bile ducts,* has led to renewed experimentation directed toward solving recurrent problems associated with the postcholecystectomy syndrome. However, the significant anatomy of the region has never been worked out in some of the species under investigation. Thus the experimentalist may be unaware of the best approaches to the problem and uncertain as to the application of his findings to the human subject. Even when one discovers a fundamental difference in the reactions of species, as in the response of the feline and human biliary tracts to section of nerves, a satisfactory understanding may still depend upon recognition of anatomic differences. For these reasons, and to obtain a more comprehensive picture of the relationship of the bile duct to the extrinsic and intrinsic musculature which envelopes it, I have undertaken a comparative study of the minute anatomy of the choledochoduodenal junction in man and in a series of laboratory animals.

The first study of the animal series is based on 2 fetal specimens of the chimpanzee.[5] Except for minor differences the junctional region was found to resemble that of the human fetuses described by Schwegler and Boyden.[19]

The second study[10] deals with the dog, the animal most used in analyzing experimentally the activities of the gastrointestinal tract. In this species the intramural pathway of the bile duct is divided into a proximal infundibular portion, in which the duct traverses a long, flattened tunnel in the circular muscle layer of the gut, and a distal submucosal portion which is surrounded by a series of isolated loops of the musculus proprius. While this arrangement offers an ideal opportunity for the experimentalist to distinguish between the action of intrinsic and extrinsic compressors of the duct, the arrangement is quite different from that in man, for the human duct passes through a slit rather than a funnel in the musculature of the gut, and the musculus proprius of the human duct is not made up of U-shaped fibers binding the duct closely to the gut wall, but instead consists of an investing sheath of fibers which extends out into an erectile papilla.

The third and present study deals with the domestic cat. Theoretically this animal would seem to offer more direct comparison with man than would the dog, since in the cat and sometimes in man bile and ventral pancreatic ducts open into a well-developed ampulla.

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*Sodium iodipamide.[11, 21]

MATERIALS AND METHODS

This account is based upon serial sections of the duodenum in 2 four-week-old kittens and upon macerated preparations from 9 half-grown cats. The latter preparations were obtained by immersing the first portion of the duodenum in 20 per cent nitric acid for approximately 42 hours, then dissecting away the tunica mucosa, then placing the preparations in 20 per cent formalin for hardening and for further dissection. In addition to these specimens x-rays of experimental animals used in earlier studies were examined for identification of portions of the pars intestinalis of the bile duct (Fig. 3). Finally, the results of previous experiments on the sectioning of nerves were reviewed in the light of the present findings.

THE GROSS ARRANGEMENT OF DUCTS IN THE CAT

In this species bile and ventral pancreatic ducts enter the intestine through a window on the right lateral side of the duodenum about 1.5 cm. below the pylorus (Fig. 4). Since the duodenum of the cat is not retroperitoneal one would have to swing this loop of bowel to the viewer's left in order to make it assume the position found in man. Then the bile duct would enter the duodenum on its dorsal side. Even so, the orifice of the duct would lie nearer the pylorus than in man.

The ventral pancreatic duct (of Wirsung) is the main one and is formed by the union of splenic and duodenal divisions. The duct of the dorsal pancreas is smaller and anastomoses freely with branches of the ventral duct.

The choledochal fenestra is a slitlike opening in the muscle of the gut. The slit may be parallel to the long axis of the duodenum (Fig. 6), lie at right angles to it (Fig. 7), or be placed obliquely. Its free margin is formed by the circular muscle layer of the gut (Margo ext., Figs. 7 and 13). The free margin of the longitudinal layer (hiatus, Fig. 13) is some distance from the slit. In this respect the pattern somewhat resembles that in man, where the bile duct enters a "gridiron" aperture in the muscle consisting of a lengthwise hiatus in the longitudinal layer superimposed upon a transverse fenestra in the circular layer.[19, II] However, in the cat, the margins of the aperture are concealed by the tunica serosa and further obscured, on the pancreatic side, by a blending of longitudinal and circular layers.

The internal aperture in the muscle of the gut is shown in Fig. 5 (Margo int.). In this specimen the mucosa has been removed following maceration of the gut in 20 per cent nitric acid for 42 hours. A profile of another specimen (Fig. 9) shows that the intramural passage is divided into proximal and distal portions, as in the dog, a portio infundibularis in which the ducts traverse a funnel in the circular muscle of the gut and a portio submucosa in which the ampulla of Vater traverses the tela submucosa on its way to the papillary orifice.

The point of entrance of the two ducts into the ampulla is not easily established. In the embryo, one may assume that the ducts unite to form a simple tube, the hepatopancreatic duct.[19, I] During fetal life, however, the

wall of this tube becomes folded in such a way as to form a series of telescoping funnel-shaped chambers, the whole comprising the ampulla of Vater.* Peripherally the duct is characterized by sacculations and internally by a series of valves pointing toward the orifice (Fig. 14). The pattern is not unlike that in the ampulla of the human fetus.[19, I, p. 467, and III, p. 215] Yet in the cat the process is elaborated to such a degree that Gage[12] "found [it] impossible to determine in transverse sections [of an adult cat] whether a given

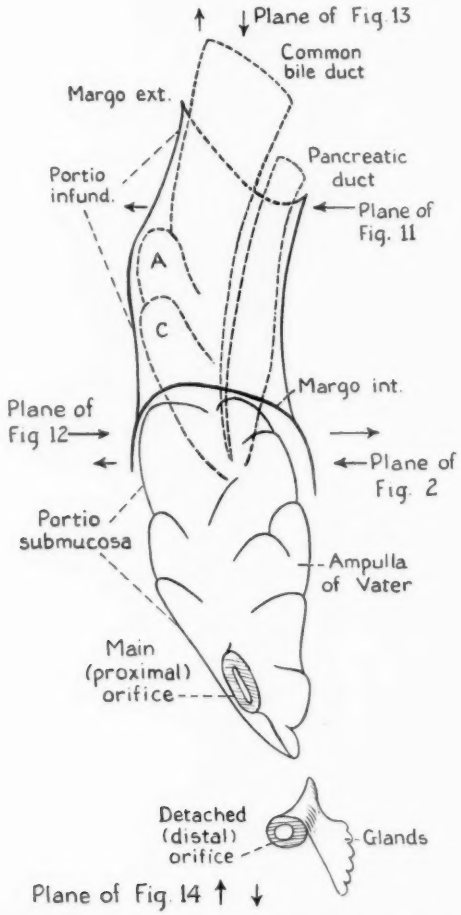


Fig 1.—Graphic reconstruction of the pars intestinalis of the common bile duct of a 4-week-old kitten, made from longitudinal sections (×15). Two of these are shown in Figs. 13 and 14, and their planes are indicated in Fig. 1. For convenience, the approximate positions of transverse sections used to illustrate this paper are also indicated (Figs. 2, 11, and 12). Note division of region into the portio infundibularis, a muscular funnel traversed by both ducts, and the portio submucosa, occupied chiefly by the ampulla of Vater. See text for discussion of double orifice at ampulla.

part belonged to the ductus choledochus, the duct of Wirsung, or the ampulla proper." Accordingly, he resorted to colored injections, filling first one duct and then the other. From these preparations he concluded that the bile duct opens into the ampulla close to the intestinal orifice, but that the pancreatic

*It is not without significance that Abraham Vater's own name for this structure was "tubercle" or "diverticulum."[4] In sooth, it is a nodular structure made up of many diverticula.

duct empties much more proximally. However, sections of the duodenum in kittens lead me to conclude that both ducts open together into the proximal portion of the ampulla.

Fig. 1 represents a reconstruction made from sagittal sections of a kitten 4 weeks old. The orientation is the same as that of Fig. 5; that is, the ampulla and ducts are represented as seen from the inside of the gut, as if through transparent walls. The margo internus is indicated as being in the plane nearest to the observer and the margo externus (heavy dash line) as farthest away. Above, between outer and inner walls of the muscular funnel, run the ducts. Below, the ducts terminate deep in the interior of the ampulla near its base.

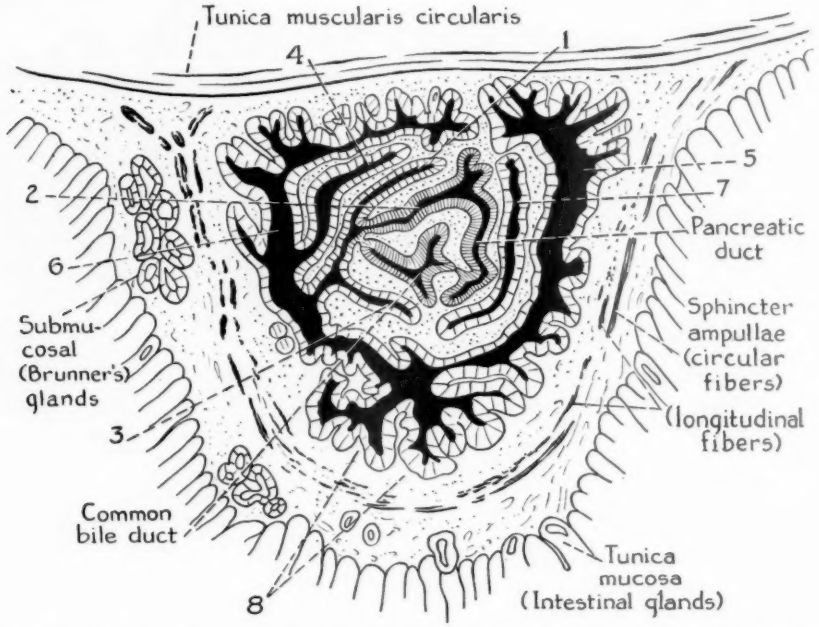


Fig. 2.—Tracing of transverse section (No. 468) of the portio submucosa of a 4-week-old kitten, at level shown in Fig. 1. Here, bile and pancreatic ducts are seen emptying into the chamber of the ampulla of Vater. This chamber has given rise to a series of recurrent, concentrically arranged saccules, the inner walls of which form valves pointing into the lumen and toward the orifice (Fig. 14). At this level, 8 saccules are present (1-8). At a more proximal level (Fig. 12) only the inner ones are present (1-4).

The complexity of this pattern is illustrated by a transverse section through a second kitten at the level of the opening of the ducts (Fig. 2). At this point the bile duct has tapered from a wide channel with recurrent saccules to a narrow, pleated tube which is opening on one side into the pancreatic duct and on the other side into a recurrent saccule of the ampulla (Saccule 2). These openings are 56 sections (half a millimeter) below the place where the pancreatic duct first widens into Saccule 2.

At the level shown in Fig. 2 there are 8 recurrent saccules that have originated from the ampulla and that lie concentrically around the terminations of the two ducts. These saccules are numbered in the order in which they appear as one follows the sections toward the orifice of the ampulla. Similarly, the bile duct gives rise to retrograde saccules. Three such diverticula could

be identified in the transverse series, the lowest of which is designated "C" in Fig. 12. In the sagittal series it would have been difficult to distinguish ampullary from biliary saccules without the knowledge provided by the transverse series. With this aid saccules A and C (Fig. 1) have been identified as originating from the bile duct.

Another striking feature is the marked tapering of the common bile duct as it enters the ampulla (Fig. 2). At this level the pancreatic duct is wider than the bile duct, but proximally the bile duct is the larger of the two.* A similar situation is present in the human fetus[19, III] (Fig. 17); but it is even more striking in the chimpanzee fetus[5] (Figs. 7 and 8). This tapering of the bile duct into a nozzle, coupled with the presence of retrograde saccules, would seem to prevent reflux of intestinal contents into the bile duct, but not reflux of bile into the pancreatic duct.

The last variant in the arrangement of ducts is duplication of the orifice of the ampulla. Fig. 1 shows a specimen having two openings into the intestine, although the distal one has lost its connection with the main cavity of the ampulla. Doubtless such duplication is rare in full-grown cats. However, it should be recalled that in the human fetus the ampulla normally has proximal and distal openings in early stages.[19, I] Both of these are said to persist in 1 per cent of adults.

THE MUSCULATURE OF THE INTRAMURAL PASSAGE

This study is based not only upon serial sections of the 2 kittens described above but upon macerated preparations from 9 full-grown cats.

The Portio Infundibularis.—The funnel portion of the intramural passage is that which extends from the margo externus to the margo internus (Figs. 9 and 13). Its length in formalin specimens, as measured from the middle of one margin to the middle of the other, averages 3 mm. (with a range of 2.5 to 4.0 mm.). It is shortest in cats with a longitudinally placed choledochal fenestra (Figs. 6 and 10). In such specimens the course of the pancreatic duct through the funnel is greatly shortened, but that of the bile duct is of usual length. When the funnel is sectioned longitudinally (Figs. 8 and 10) its most characteristic feature is seen to be the interductual septum which separates the funnel into two compartments, one for each duct. This muscular septum begins at the external orifice (Fig. 6) and extends half to two-thirds of the way through the funnel (Figs. 8 and 10). As seen in transverse sections the septum originates in muscular trabeculae lining the wall of the funnel on the side away from the mucosa (Trab., Fig. 11), then curves around the bile duct to join fibers lining the funnel on the side nearest the mucosa (*, Fig. 11). Contraction of the septum would therefore compress the bile duct. Frequently, also, the septum sends fibers in the opposite direction to partly enclose the pancreatic duct (Fig. 8). But these are minor strands. More significant is the fact that the presence of trabeculae inside the funnel greatly

*In Fig. 1, the pancreatic duct also seems to taper, but this is an illusion due to the fact that it is seen edgewise (Fig. 2).

diminishes the diameter of the passages (Fig. 13). Putting all these facts together it is difficult to see how the septum could operate independently of the funnel, which is derived from the circular muscle layer of the gut.*

In addition to the septum and its derivatives there is a delicate network of muscle strands immediately surrounding the bile duct, suggestive of a muscularis mucosae. These fibers are so intimately related to the mucosa of the duct that they presumably adhere to the duct when it is dissected away from the funnel.

The Portio Submucosa.—The distal portion of the intramural passage is occupied chiefly by the ampulla of Vater. From margo internus to the farther edge of the papillary orifice it measures, on the average, about 4.3 mm., with a range of 3 to 6 mm. Throughout its length it is surrounded by a delicate sheath of interlacing muscle fibers (Fig. 5). This is composed of two thin layers, the outermost of which is formed by U-shaped fibers which loop backward to enclose the ampulla (Fig. 9). Each end of a "U-fiber" is attached to the circular muscle of the gut (Figs. 2 and 12). In midsagittal section these fibers seem to be a continuation of the margo internus (Musc. propr. circ., Fig. 14) but a side view of the papilla reveals their true origin (Fig. 8) and the fact that the more proximal ones arise from the interductual septum within the funnel.

The innermost layer of the sheath is composed of longitudinally and obliquely running fibers (Musc. propr., Fig. 8). These are tapered continuations of longitudinal trabeculae lining the funnel. (See X, Figs. 5 and 10 and Musc. propr. long., Fig. 14.) Their origin is well displayed in tangential sections, where they can be seen spraying out from beneath the margo internus.

This double-layered sheath of fibers corresponds superficially to the sheath enclosing the portio submucosa of the dog. In that species, however, there is no inner longitudinal layer, and the sheath is made up of obliquely placed bands that could exert considerable pressure on the distal portion of the bile duct. Moreover, the dog has a terminal sphincter which encloses both ducts at their orifice. In the cat, however, contraction of the fibers around the ampulla could be expected only to shorten and compress the saccules of the ampulla in emptying. This contraction would impede the flow of bile and pancreatic juice only slightly even though the proximal portion of the sheath surrounds the ampulla at the level where the ducts terminate (Fig. 2). Consequently any selective regulation of either bile or pancreatic outflow must take place in the funnel. Here, however, the fibers compressing the bile duct belong to an interductual septum which is a part of the circular muscle layer of the gut. Thus one encounters, again, an arrangement of fibers which is different from that of any other species so far studied.

*The American anatomist, Gage, described this "wedge of muscular fibers between the ducts" in 1879, thus antedating Oddi. He stated that the wedge "was made up partly of longitudinal and partly of circular fibers. Near the external surface of the longitudinal layer, some of the fibers are sent straight between the ducts, and other apparently special fibers wind round the two ducts and form a common sphincter. Still other fibers pass around the ducts separately, and thus form special sphincters. The one which belongs to the duct of Wirsung is most marked."

DISCUSSION

The purpose of these morphologic studies has been to contribute to an understanding of the functional mechanisms of the extrahepatic biliary tract. Presumably no one would dispute the view that in laboratory mammals (as perhaps contrasted with man) motor activities of the tract are subject to both nervous and hormonal control. The problem has been, "What nerves, and to what extent do nerves control these movements?"

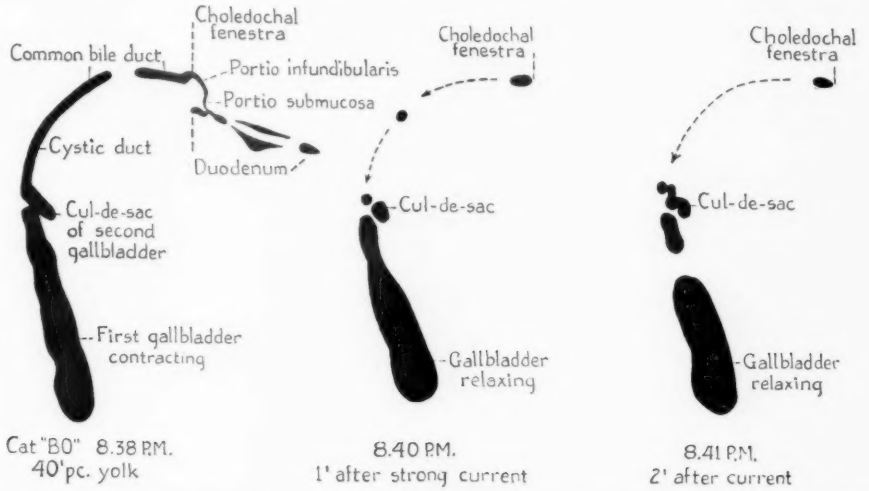


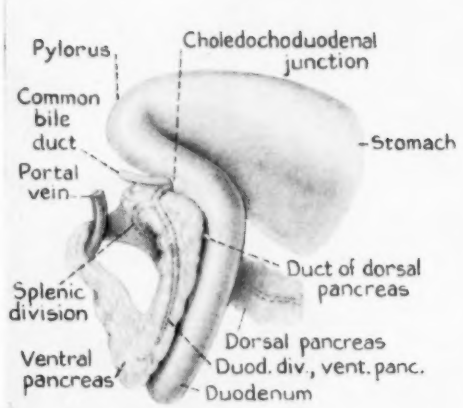
Fig. 3.—Enlarged tracings of a series of x-rays of a living cat showing the hitherto unrecognized appearance of the pars intestinalis of the bile duct when visualized with Lipiodol. These figures illustrate reflex inhibition of the gall bladder induced by faradic stimulation of the pyloric limb of the stomach. Laparotomy was performed under anesthesia in the morning. The gall-bladder bile was replaced by Lipiodol injected through the fundus; a shielded electrode was sewn to the stomach; enameled wires were brought out through a rubber tube; and the body cavity was closed. That same night, after the cat had recovered from the anesthetic, the animal was placed purring on the x-ray table and fed egg-yolk. Note elongation and emptying of gall bladder (Fig. 1). Subsequently contraction of the stomach was induced by a tetanizing current applied to the electrodes in the gut. Within another minute (often within 30 seconds) the gall bladder shadow had widened and the iodized oil had dropped back into the dependent portions of the gall bladder and failed to fill it (Fig. 2). Two minutes after the current the gall bladder was almost completely relaxed (Fig. 3). This process could be repeated as desired.[3]

In my earlier experimental studies with cats the following information was obtained:

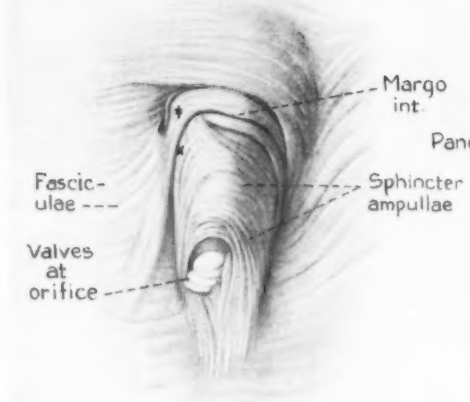
First, in a physiologically intact, unanesthetized animal contraction and emptying of the gall bladder may be inhibited promptly by applying an induction current to any part of the gut.[3] One such experiment, reproduced in Fig. 3, is especially pertinent since here is pictured, for the first time in the living cat, the two intestinal segments of the bile duct. (For details of experiment, see legend of Fig. 3.)

Second, section of the splanchnic roots of the celiac ganglia prevents the inhibitory effects noted above.[14] From this it is inferred that the sympathetic pathway inhibits contraction of the gall bladder. Its opposite action on the sphincter may be presumed, but has not been demonstrated.

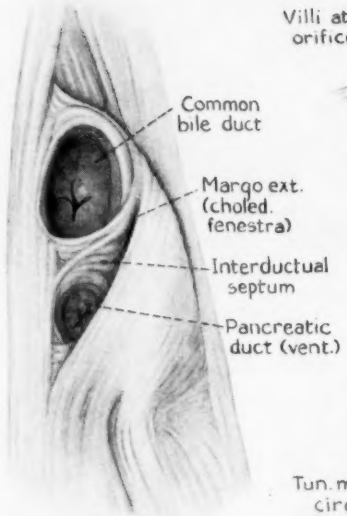
Third, the rate of emptying of the gall bladder is not affected appreciably by sectioning the splanchnic nerves. It can be retarded somewhat by sectioning the gastroduodenal nerve to the sphincter region (the fibers of which are



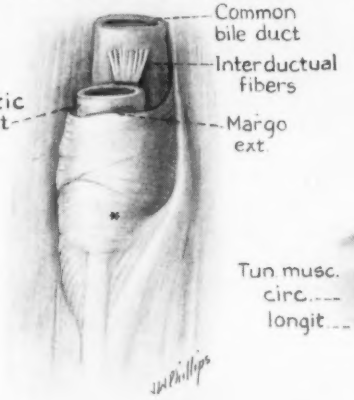
4. GROSS RELATIONS



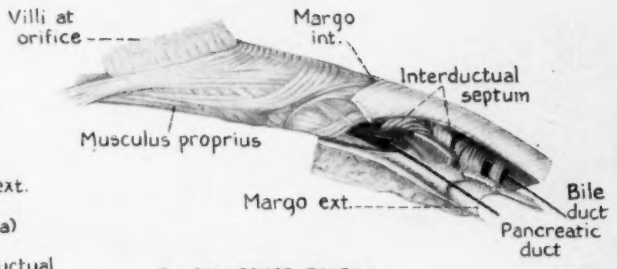
5. SUBMUCOSAL ASPECT



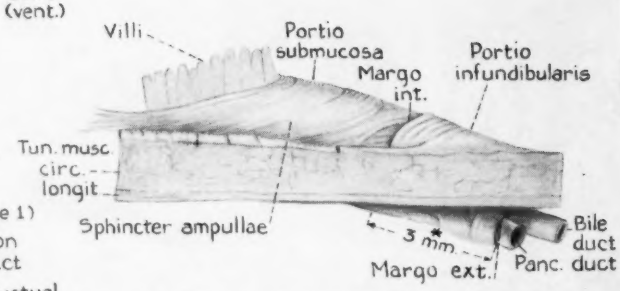
6. CHOLEDOCHAL FENESTRA (type 1)



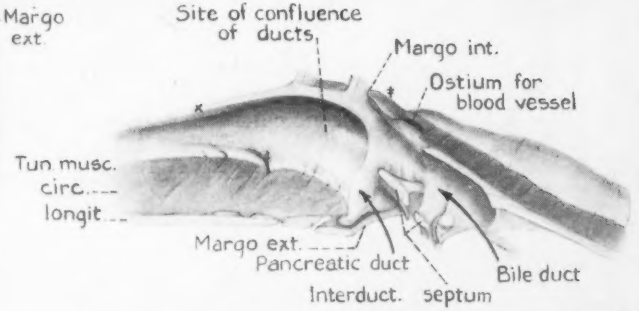
7. CHOLEDOCHAL FENESTRA (type 2)



8. DISSECTED PROFILE



9. PROFILE OF INTRAMURAL PATHWAY



10. DISSECTED PROFILE

Figs. 4-10.—Sketches by the author of macerated preparations of the pars intestinalis of the cat (×5, except Fig. 4). (Prepared for publication by Miss Jessie W. Phillips.)

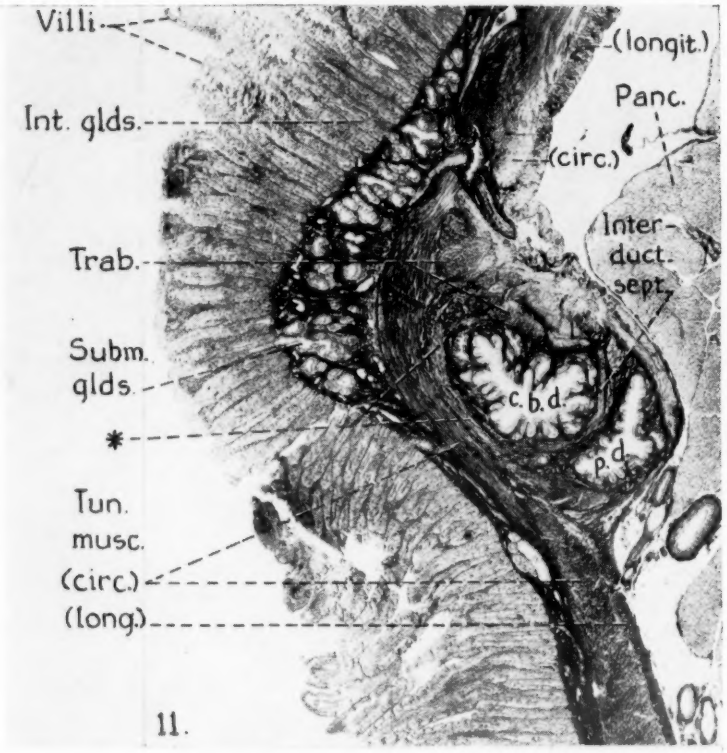
Fig. 4.—Sketch of the duodenum and associated ducts as they appear after laparotomy. When the animal is placed on its right side for roentgenography, the duodenum assumes a dorsal posture more nearly resembling that of man (Fig. 3).

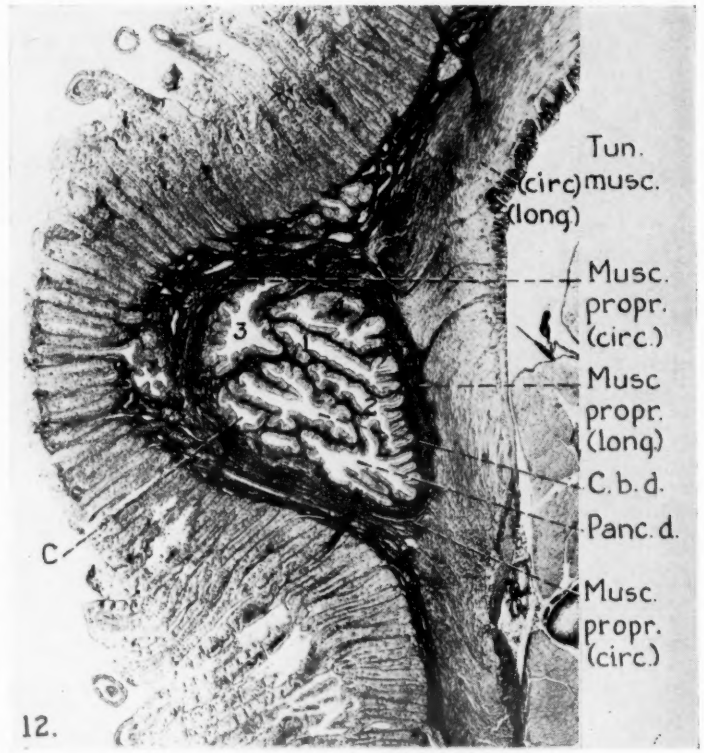
Fig. 5.—Papilla of Cat No. 7 viewed from the inside of the duodenum after maceration and removal of the tunica mucosa. Subsequently, the specimen was bisected lengthwise and the half to the viewer's left sketched from the cut side (Fig. 10). The double dagger in both figures indicates the bundle of circular muscle which forms the distal end of the funnel. The sessile papilla is ensheathed by a thin, double-layered musculus proprius, the sphincter ampullae. As the papilla emerges from the margo internus it receives strands of longitudinal fibers from trabeculae lining the inner wall of the funnel (see X, Figs. 5 and 10; Musc. propr., Fig. 8; and Trabec., Fig. 13). Fasciculae, bundles of circular muscle forming outer wall of funnel.

Figs. 6 and 7.—Sketches of the choledochal fenestra in Cats Nos. 8 and 9. These picture the "window" through which bile duct and ventral pancreatic duct enter the funnel in the intestinal muscle. The free edge of the window is the margo externus. In the example shown in Fig. 6, the interductual septum is visible in the window.

Figs. 8 and 9.—Profile and dissected profile of the specimen shown in Fig. 7 (Cat No. 9). In Fig. 9 the specimen is drawn as it appears before dissection. In Fig. 8 the funnel has been opened and the fibers of the ampulla have been cleaned of intervening connective tissue to show their direction. The asterisk indicates identical spots in Figs. 7 and 9. The musculus proprius of the ampulla consists of two thin layers, an outer obliquely circular layer and an inner longitudinal layer (musc. prop., Fig. 8). Note that the latter is an extension of trabeculae lining the funnel. The interductual septum arises from the abmucosal side of the funnel and spreads around both ducts in this specimen. On the pancreatic side its fibers continue to the ampulla.

Fig. 10.—View of one half of the specimen pictured in Fig. 5, after bisection of papilla. Double dagger and cross indicate parts similarly designated in Fig. 5. In this view the septum can be seen arising from trabeculae that line the funnel (Trab., Fig. 11). Other trabeculae taper off into longitudinal strands of the sheath that encloses the ampulla (see X, Figs. 5 and 10).

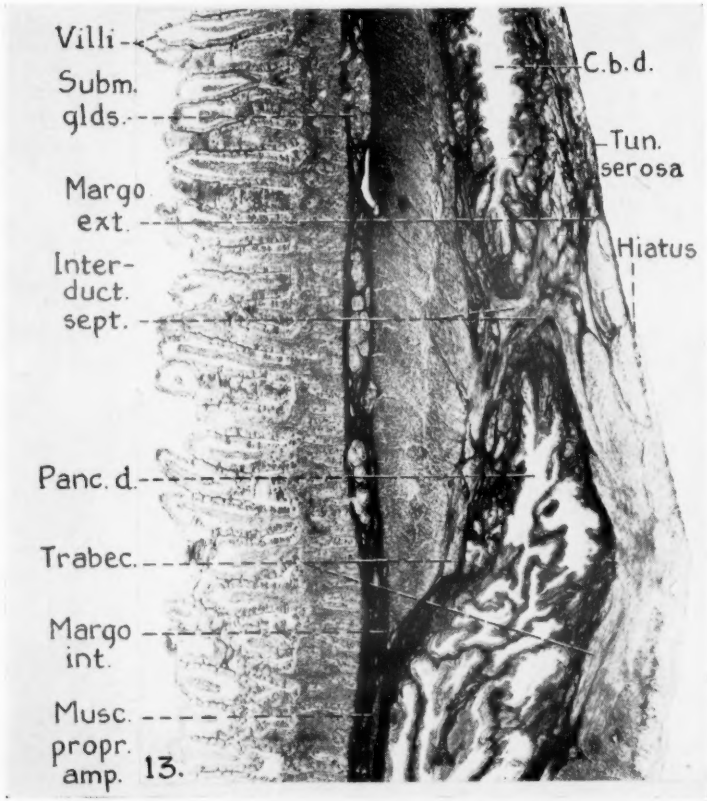




Figs. 11-14.—Photomicrographs of choledochoduodenal junction in two 4-week-old kittens. (Transverse series, ×28; sagittal series, ×26; stain, Heidenhain-Azan.)

Fig. 11.—Transverse section (No. 237) at beginning of portio infundibularis. The margo externus is just closing over the two ducts. (For orientation, see Figs. 1 and 6.) Note: (1) The interductual septum and (2) the muscle column (*) which, in later sections, connects the septum with trabeculae (Trab.) that line the funnel. Together, these structures form a special sphincter around the common bile duct.

Fig. 12.—Transverse section (No. 415) just above the junction of bile and pancreatic ducts (for orientation, see Fig. 1). At this level the ducts are surrounded by 5 saccules; Nos. 1 to 4 are diverticula of the ampulla which join it distally (Fig. 2). The fifth saccule (marked C) is a recurrent diverticulum of the common bile duct. Compare the masculus proprius of the ampulla with that in Figs. 2 and 14.



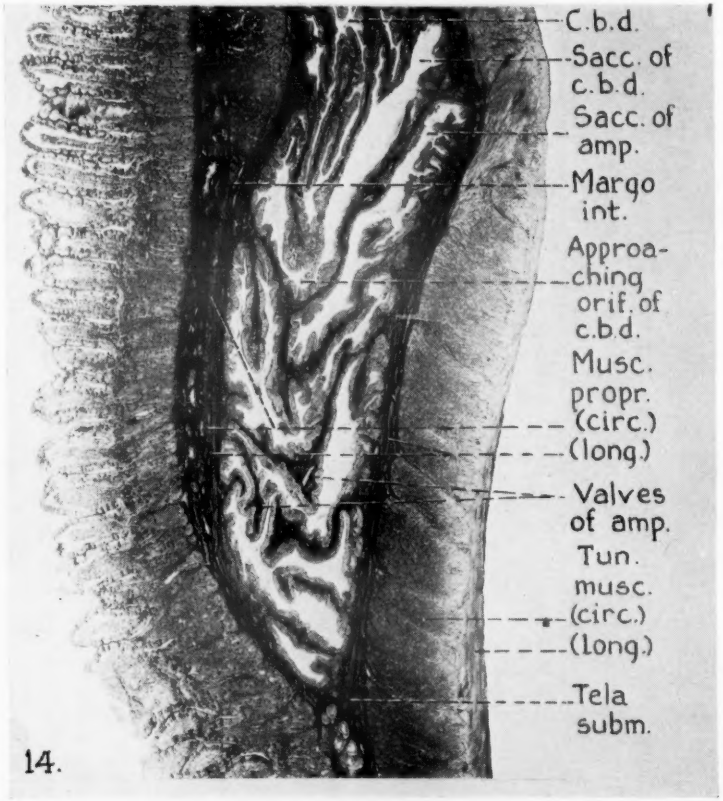


Fig. 13.—Sagittal section No. 67. (For orientation, see Fig. 1.) Here, the interductual septum is cut obliquely. Note double nature of septum (Fig. 8) and its origin from trabeculae lining the funnel. The bile duct is passing deep to the septum; the pancreatic duct passes on the side toward the observer. Note how the septal fibers extend out along the bile duct and thus appear in the fenestra (Figs. 6 and 7). The longitudinal muscle of the gut fails to reach the margo externus (see hiatus, Fig. 13.) However, both are covered by the tunica serosa.

Fig. 14.—Sagittal section No. 99 (Fig. 1). This is another tangential section of the ampulla, showing overlapping V-shaped arrangement of saccules. Deep to this section the points of the V's join the main lumen of the ampulla. The uppermost "V" is formed by saccules of the common bile duct. Note the thin, double-layered musculus proprius of the ampulla. The outer circular layer appears on the mucosal side only, since its fibers are U-shaped loops (Fig. 8).

largely derived from the right vagus) or retarded more by sectioning the left vagus below the diaphragm or retarded still more by sectioning the right vagus. Cats deprived of the right vagus require three times as long as the controls to empty half the contents of the gall bladder (219 minutes as against 70 minutes). From this it is inferred that the right vagus mediates contraction of the gall bladder and relaxation of the sphincter apparatus.

Fourth, section of both vagi (12 cases) or both vagi and splanchnics (11 cases) slightly increases the rate of emptying over that of the controls (12 cases). The time required to empty the gall bladder half way is only 64 minutes and 68 minutes, respectively, as compared with the 70 minutes of the controls.[9] An explanation for this is found in the observation by Meek and Herrin[18] that section of one vagus in the cat does not affect the rate of emptying of the stomach but that section of both vagi relaxes the pyloric sphincter and so decreases the initial time required for the passage of food into the duodenum. The faster production of cholecystokinin will then compensate for the loss of vagal stimulation of the biliary tract.

The present study reveals the close investment of ducts by the powerful circular muscle of the intestine and its derived interductual septum, and shows how quickly the flow of bile in the cat could be stopped by reflex contraction of the sphincter and simultaneous inhibition of the gall bladder. The presumption is that both actions are mediated by the sympathetic nervous system.* The fact that subdiaphragmatic section of the right vagus markedly retards emptying of the biliary tract in the cat would seem to indicate that the sphincters of the interductual septum fail to relax sufficiently after the nerve is cut. It will be recalled that in Westphal's experiments with rabbits and guinea pigs,[20] weak stimulation of the vagus contracted the gall bladder and relaxed the sphincter papillae. Possibly that portion of the interductual septum which winds around the bile duct in the cat (*, Fig. 11) is selectively innervated by the right vagus or has a different threshold of stimulation. Or possibly the formation of cholecystokinin is blocked by severance of the right vagus. Recently it has been shown by Hong, Magee, and Crewdson[13] that intravenous administration of a ganglionic blocking agent (hexamethionum) prevents the formation of cholecystokinin in dogs.

In conclusion, it may be stated that the mechanism which regulates the flow of bile in man differs from that of the cat in at least five respects: first, it has been demonstrated by experiment that in man the sphincter apparatus can act independently of the duodenal muscualture[1]; second, faradic stimulation of stomach and duodenum (through swallowed electrodes) does not inhibit discharge of bile, either reflexly or otherwise[7]; third, partial or complete section of the vagi in man does not retard emptying of the biliary tract[15]; fourth, in man the tract does not react to neuromimetic drugs[2]; and fifth, magnesium sulfate relaxes the sphincter and induces the first phase of emptying of the gall bladder in man[6] but has no such effect in cats.[8]

*This is based on the experiments noted above. But it is interesting to find in the sheep and ox that adrenalin causes contraction of the sphincter of Oddi while atropine relaxes the duodenal musculature.[17]

SUMMARY

This is the third of a recent series of studies comparing the choledochoduodenal junction in man with that of laboratory mammals. Previous papers have dealt with the chimpanzee and the dog. This paper is concerned with the cat.

In the cat, as in the dog, the intramural course of the bile duct may be divided into two portions, a proximal portion which passes through a funnel in the duodenal musculature and a distal portion which traverses the tela submucosa. But here the resemblance ends, for proximally the bile duct of the cat is accompanied by the pancreatic duct (of Wirsung) and distally it joins that duct to form the ampulla of Vater.

Furthermore, bile and pancreatic ducts in the cat are separated by a specialized structure, the interductual septum which is a part of the funnel musculature. Moreover, the musculus proprius of the ampulla of the cat is relatively weak, while the corresponding distal segment in the dog is encircled by strong muscular loops. The human pattern differs from both animals in that the bile and pancreatic ducts enter the duodenum through a slit in the intestinal muscle and thus are not subject to the pressures of a muscular funnel.

It is pointed out that physiologically the sphincter mechanism in man differs from that of the cat in several respects which are enumerated and discussed.

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A VERSATILE ANESTHESIA MACHINE FOR EXPERIMENTAL SURGERY

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INTRODUCTION

LABORATORIES for research in cardiovascular physiology or experimental surgery frequently have occasion to carry out open chest experiments or thoracic surgical procedures where carefully controlled intermittent positive pressure or alternating positive-negative pressure is essential. Where animals of a wide range of sizes are employed, it is important to have an anesthesia machine adaptable enough to permit its use with animals ranging from cats or rabbits up to and including large dogs.

Furthermore, since few laboratories feature extensive facilities for development and maintenance, it is important to choose a design which is simple to construct and requires little or no special attention, yet is dependable and readily adjustable to allow for different animal size.

DESCRIPTION

An anesthesia machine meeting these requirements has been designed and successfully used at the Aero Medical Laboratory. The essential components are shown in Fig. 1. The machine features a closed system, with inlets for three or four different anesthesia gases as desired, or diethyl ether may be added under pressure from an atomizer or pump. The gases pass through a one-way valve to enter the endotracheal tube and are exhaled into the exhaust circuit through another one-way valve. A soda lime cylinder absorbs carbon dioxide, and the gas mixture again enters the inhalation side of the circuit.

The pressure source for the system is supplied by the usual type anesthesia bag enclosed in a plexiglass globe or the commercially available Seeler assistor.* This bag can be manipulated by hand if desired, but is more satisfactorily operated by inserting a Seeler resuscitator in the inlet to the globe and accomplishing the pressure changes by connecting this to a steady source of compressed air of 35 mm. Hg pressure. A small compressor, vacuum cleaner, or tank of compressed air are equally suitable as pressure sources.

The Seeler resuscitator features a choice of positive pressures up to 16 mm. Hg and negative pressures to 8 mm. Hg. With this arrangement it is possible to select alternating positive-negative phases or maintain intermittent

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*The Seeler respirator and Seeler resuscitator are manufactured by Globe Industries, Inc., Dayton, Ohio.

positive pressure respiration according to needs. This is an advantage not shared by most other resuscitators. The rates of flow for the oxygen-gas mixture are adjustable and vary between 0 and 21 L. per minute.

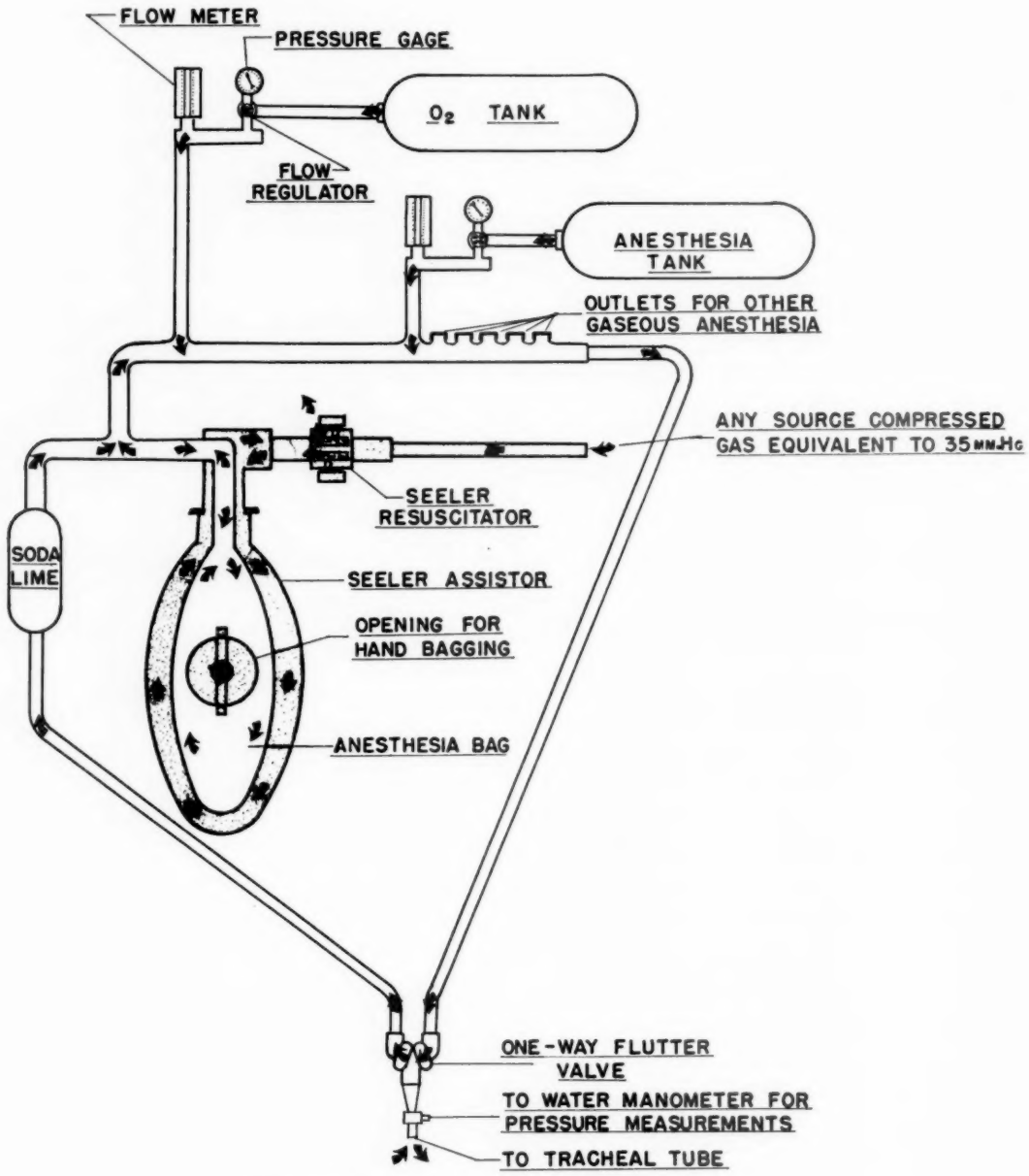

Fig. 1.—Diagrammatic layout of anesthesia unit.

Another adaptation that is useful when intravenous anesthesia is employed is the use of the Seeler resuscitator alone. Under these conditions the resuscitator is activated by a source of compressed air or oxygen, with its flow regulated to 35 mm. Hg by a reduction valve. The resuscitator is connected to the

compressed oxygen source on one hand and directly to the indwelling cuffed endotracheal tube on the other. This affords the same choice of intermittent positive pressure or alternating positive-negative pressures as described above for inhalation anesthesia. In addition, continuous positive pressure may be used for inflation of the lungs at intervals; or continuous negative pressure for short intervals may be used for aspiration.

Maintenance is no problem with the Seeler resuscitator. The control of this device is regulated by small permanent magnets, and no electrical connections or aneroid systems are involved. One such resuscitator was in constant use in our laboratory over a period of 5 years. This was recently checked for calibration and was found to have maintained perfect accuracy.

This anesthesia machine is proposed for experimental surgical laboratories. However, several clinical applications of the Seeler resuscitator with an oxygen source also come to mind. These include such diversified problems as poliomyelitis patients with pulmonary edema, where intermittent positive pressure may be lifesaving. Another application is for patients with crushing injuries of the chest, where their condition is too precarious to permit external fixation. In this situation intermittent positive pressure respiration can be maintained over long periods when combined with tracheotomy. There are also situations occurring postoperatively where prolonged intermittent positive pressure or alternating positive-negative pressure must be maintained for fairly long periods. The latter feature can be easily obtained and regulated by this device, thus representing a considerable advantage over hand-operated "bag breathing." The feature of having continuous negative pressure when desired also makes available a ready source for emergency suction.

We wish to thank Mr. Henry Seeler, Engineering and Development Branch, Aero Medical Laboratory, for his help in adapting this system to small animal use.

POST-PARTUM FISTULA-IN-ANO: A COMPLICATION OF EPISIOTOMY

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THE natural history and pathologic anatomy of the usual type of fistula-in-ano have been well described and adequately documented in several recent, excellent articles.[1, 2, 4] It is not the purpose of this paper to review that topic. Most papers dealing with the subject, however, have failed to emphasize or even mention a most distressing type of "man-made" fistula, namely, that which may develop in the episiotomy wound at some time in the post-partum period. It is important that this type of fistula-in-ano be given special attention and discussion for we are seeing it clinically with increasing frequency. The process is not being recognized during a stage in its development when definite measures could be undertaken to return the wound to its normal state of healing. Later recognition is more difficult and the task of repair is considerably more complicated.

The common type of fistula-in-ano is a natural disease, but the episiotomy fistula is an artificially produced process and thus follows a more precise and anticipated pattern. The external opening of the tract is always at or very near the episiotomy scar, while the internal opening is found at any level in that quadrant of the rectal wall approximate to the episiotomy site (Fig. 1). This internal opening is usually not at the level of the mucocutaneous junction, as would be expected in the natural form of the disease, but 1 to 2 cm. above this line. The pathologic process involved in the formation of the fistula results from infection deep in the wound, originating when a suture is inadvertently passed through the wall of the bowel allowing leakage into the perirectal tissue.[3]

Generally speaking, the process may be classified into the early (acute) or the late (chronic) phase of the disease.

ACUTE FISTULA

Healing of the normal episiotomy wound occurs within five to seven days, and continued tenderness, throbbing, or sensitivity beyond a three-day period should alert the obstetrician to the possibility of complication. Outward appearance of the incision may be misleading, for there is likely to be edema, congestion, and superficial infection in every perineum soon after delivery. A low-grade fever and leukocytosis may reflect deeper infection, and dysuria or rectal tenesmus indicate irritability of the pelvic structures. These symp-

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toms were typical in fourteen cases of episiotomy fistula-in-ano which we have observed. Without exception, the women gave a history of persistent severe pain in the episiotomy wound for two to four weeks after delivery. Most of the patients suffered to the point of not being able to sit down for any length of time and, for a period of from seven to ten days after hospital discharge, were forced to eat their meals while standing.

When local symptoms persist beyond three or four days, therefore, it behooves the attending obstetrician to place a finger gently into the rectum and inspect the wall of the bowel approximate to the healing tissue. Evidence of adjacent infection or perforating sutures can be palpated and the diagnosis established. In the former instance, opening a portion of the wound to promote drainage should be undertaken. In the latter case, immediate removal of the perforating strand of suture through an anoscope must be accomplished. The patient is then placed on antibiotic therapy, and the local application of hot compresses is given as adjunctive treatment. Within a few days the wound should be well on its way to normal recovery.

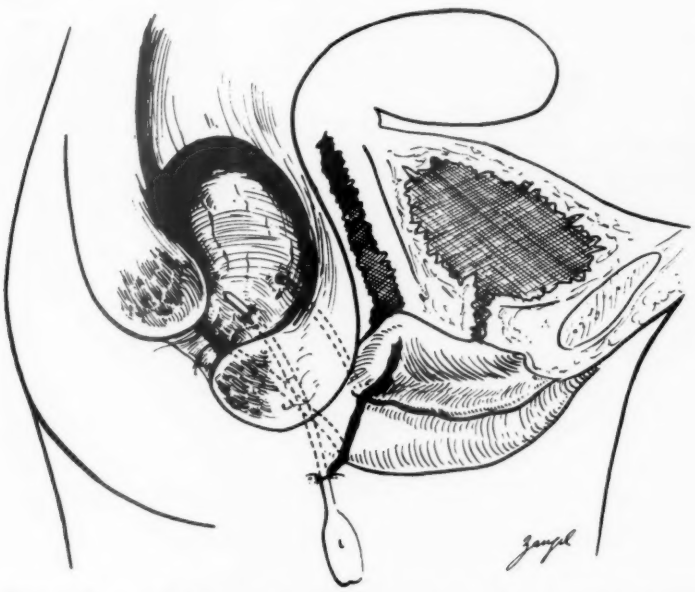

Fig. 1.—Cross section of the female perineal structures showing the possible locations of episiotomy fistula-in-ano. The internal opening is usually above the mucocutaneous line and little or no rectal discomfort is experienced by the patient.

CHRONIC FISTULA

When the acute phase of the episiotomy fistula-in-ano goes unnoticed and the patient leaves the hospital with an unusually painful perineum, the period of chronicity will depend upon the degree of infection, the virulence of the organisms, and the tolerance of the patient. It is surprising to observe that even after the acute symptoms have subsided, many women admit to a long period of discomfort in the perineum, a draining episiotomy scar and the inability to have normal intercourse following the birth of their child. While it is evident by its relative infrequency that all of these women do not develop

fistula-in-ano, such an observation does indicate that many of these wounds are infected despite excellent obstetrical technique. Symptoms of chronic post-partum fistula-in-ano are thereby often masked by complaints that the obstetrician hears quite frequently. Because of the fact that the internal opening of the fistulous tract is usually above the sensory fibers ending at the mucocutaneous junction, the chronic episiotomy fistula is rarely accompanied by rectal pain. The external opening, however, is frequently misdiagnosed as episiotomy abscess, infected Bartholin's cyst, comedo-nevus or infected sebaceous cyst, and local therapy is directed at its removal. This invariably leads to further infection, scarring and distortion of the tissue, and the draining fistula, of course, recurs in a short time, much to the annoyance of both patient and surgeon. In the fourteen cases of post-partum fistula-in-ano which we have observed in the past eight years, the longest period of time elapsing before correct diagnosis was made occurred in a woman whose only child was born nine years prior to having the fistula repaired. This woman was seen because of an acute and severe perineal cellulitis following the surgical removal of what was thought to be an infected Bartholin's cyst (Fig. 2).

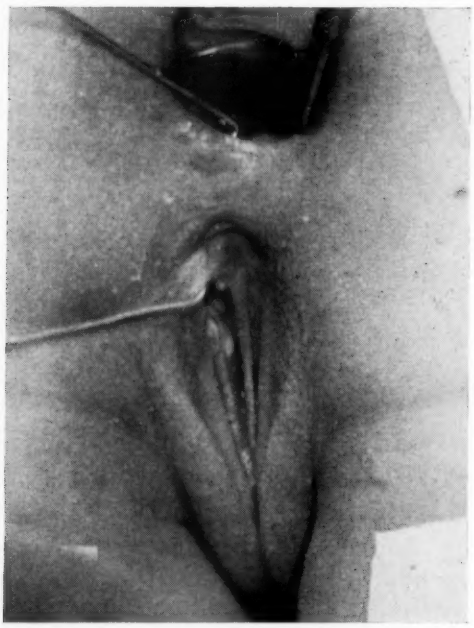

Fig. 2.—This patient's only child was delivered nine years prior to surgical removal of what was diagnosed as infected Bartholin cyst. The wound failed to heal and a severe pelvic cellulitis developed. The two probes indicate the internal and external openings of the fistulous tract. Considerable scarring and distortion of the external opening developed as a result of the infection.

Chronic cases of post-partum anal fistula are surgically corrected according to the same principles which govern the repair of the usual forms of fistula-in-ano. The tract must be removed or uncovered in its entirety with assurance that the internal opening is identified and eliminated. Depending on the depth of the internal opening, either a one- or a two-stage procedure may be

elected. The use of the Seton is especially valuable when the internal opening is deep to the main components of the external anal sphincter[1] and the two-stage operation is the procedure of choice.

PROPHYLAXIS

Local inflammatory symptoms in the episiotomy wound undoubtedly draw the obstetrician's attention to the impending fistula in most of the cases, and immediate therapeutic measures prevent further difficulties. It has long been the routine procedure of most obstetricians to place a finger into the rectum during the episiotomy closure in order to feel the needle point if it should accidentally pass into the lumen of the ampulla. This maneuver, in our opinion, may actually be of more harm than it is good, for it is impossible to feel the entire lateral rectal wall as each suture is passed and only if the operator's finger were pricked by the needle could he be certain where the point actually moved. The false sense of security obtained by having carried out this traditionally important phase of the repair, however, may cloud one's perspective when evaluating the importance of local signs and symptoms in the early post-partum episiotomy wound. A far more important procedure, in fact an imperative one, that satisfactorily assures the operator that an error has not occurred, is a thorough rectal examination of the entire wall of the ampulla as the final act prior to removing the patient from the delivery room table. The use of lightweight absorbable suture material is also recommended. Heavy chromatized or nonabsorbable knots will frequently serve as the focus for stubborn infection and granulation in the wound.

These simple and common-sense measures are capable of preventing disabling post-partum pain and annoyance and the need in many instances of subsequent surgery.

SUMMARY

Post-partum fistula-in-ano results from an infection deep in the episiotomy wound. The infection originates when a suture is inadvertently passed through the wall of the rectum. When throbbing, pain, tenderness, and local signs of infection persist beyond four days after delivery, the development of an acute fistula should be suspected. Immediate removal of the offending stitch must be carried out. Later recognition of chronic fistula is difficult and the patient may undergo perineal surgery for Bartholin's cyst, sebaceous cyst, or local abscess before the true nature of the disease is suspected. It is emphasized that only by careful examination of the rectal wall as the final act in episiotomy repair can the obstetrician be certain that perforation has not taken place.

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INTRAMURAL HEMATOMA OF THE INTESTINE, A RARE CAUSE OF INTESTINAL OBSTRUCTION

Review of the Literature and Report of a Case

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A RARE consequence of nonpenetrating injury to the abdomen is intramural hematoma of the intestine, which may cause a partial or complete intestinal obstruction. The recent finding of such a lesion in a 4-year-old patient on the L. S. U. Pediatric Surgery Service of the Charity Hospital led to a search of the literature and discovery of 33 previously reported cases.

In 1838 McLauchlan[17] reported a case of fatal duodenal obstruction due to a "false aneurysm" of the first and second portions of the duodenum. This is probably the first recorded case of intestinal obstruction caused by an intramural hematoma. In 1859 Bristowe,[3] in doing an autopsy on a 23-year-old man who died of rupture of an abdominal aneurysm, found a retroperitoneal hematoma which had caused obstruction of the third portion of the duodenum. Von Khantz[28] in 1908 reported multiple hematomas of the small bowel of a hemophiliac who died shortly after exploratory laparotomy for intestinal obstruction.

The first record of a patient surviving such a lesion was in 1914 when Vogel[27] evacuated a subserosal hematoma from the sigmoid of a 16-year-old boy who had had a hernioplasty under local anesthesia several days previously; the sigmoid was supposedly injured during injection of the anesthetic. Since that time there have been infrequent reports of intestinal obstruction caused by subserosal and submucosal hematomas, usually traumatic, although the history of trauma was sometimes obtained only after surgery.

The first description of the roentgenologic findings was in 1948 by Liverud[15] who reproduced the x-rays of a 3-year-old boy with an intramural hematoma of the upper jejunum. He commented on the filling defect in the lumen and the prominent folds of mucosa demonstrated after administration of barium. The largest series to date is that of Felson and Levin[5] who reported in 1954 the typical x-ray findings in four cases of intramural hematoma of the duodenum; one of their patients was not operated on but all four survived.

Review of the literature reveals a total of 33 cases of intramural hematoma causing intestinal obstruction. The diagnosis was made at autopsy in nine patients, two of whom had been subjected to laparotomy. Of the remaining 24 patients, 23 are known to have survived. One was treated conservatively, but the

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typical x-rays leave no doubt as to the nature of the lesion. There is no correlation between the site of the lesion and the age of the patient or the nature of the trauma. The anatomic distribution of the hematomas is listed in Table I.

TABLE I

LOCATION	SURVIVED	SUCCUMBED	TOTAL
Esophagus		1	1
Duodenum	12	4	17*
Jejunum	4	1	5
Ileum	1	2	3
Cecum	2		2
Ascending colon	3		3†
Descending colon	1		1†
Sigmoid	1	1	2
Total	24	9	34*

*In one case the outcome was not reported.
†All of these hematomas were chronic lesions treated by partial colectomy weeks or years after the injury.

Of the total of 33 patients, a history of trauma was obtained in 21, including four in whom this history was elicited after operation. The trauma varied from crushing between trucks to injury sustained by a lady gardener while lifting potted chrysanthemums. In most cases the trauma consisted of a direct blow to the abdomen, but in three patients the injury resulted from puncture by a needle.

There was no history of trauma in 12 patients, including one in whom no details were reported and three children in whom the reliability of the history might be considered questionable. In two cases there was a bleeding tendency—one a hemophiliac and the other a newborn infant with an undiagnosed hemorrhagic diathesis. The hemophiliac and one additional patient complained of hiccoughs during the illness. Two patients had duodenal obstruction associated with retroperitoneal hematoma—in one the hemorrhage was apparently spontaneous, and in the other it resulted from rupture of an abdominal aneurysm. One patient had esophageal obstruction from rupture of an aortic aneurysm. One duodenal hematoma occurred as a complication of acute pancreatitis with adenocarcinoma of the pancreas. In the earliest reported case, the "false aneurysm" of the duodenum, the etiology was unknown.

Vomiting was the chief symptom produced, but of the patients with colon lesions, two complained of constipation and one of diarrhea. Most patients had abdominal pain, but this was variable, as was abdominal tenderness. Only rarely was a mass palpable preoperatively.

In general, the surgical treatment consisted of evacuation of the hematoma, supplemented by gastrojejunostomy in two cases. Five patients with lesions of the colon were subjected to resection, because of questionable malignancy or viability, and two patients had resection of a portion of jejunum or ileum for extensive hematoma.

Definitive surgery was uniformly successful; all patients subjected to evacuation or resection of the hematoma survived. One patient recovered without operation and one lived after exploratory laparotomy and intubation past the

TABLE II

CASE	YEAR	AUTHOR	AGE	SEX	SITE	ETIOLOGY	TREATMENT	RESULT
1	1838	McLauchlan	49	M	Duodenum	"False aneurysm"	Diagnosed at autopsy	Died
2	1859	Bristowe	23	M	Duodenum	Ruptured abdominal aneurysm	Diagnosed at autopsy	Died
3	1859	Bristowe	33	M	Esophagus	Ruptured aortic aneurysm	Diagnosed at autopsy	Died
4	1908	von Khantz	24	M	Ileum	Hemophilia	Exploration	Died
5	1914	Vogel	16	M	Sigmoid	Needle puncture	Evacuation	Survived
6	1915	Bastionelli	26	M	Cecum	Blow by bicycle handlebars	Partial colectomy	Survived
7	1920	Strachan	?	M	Duodenum	Crushing between trucks	Evacuation	Survived
8	1920	Stretton	22	F	Cecum	Lifting potted plants	Evacuation	Survived
9	1922	Leo	19	M	Colon	Blow in abdomen	Partial colectomy	Survived
10	1924	Braun	6	M	Ileum	?	Diagnosed at autopsy	Died
11	1928	Santoro	7	M	Jejunum	?	Evacuation	Survived
12	1929	Giordano	24	M	Colon	Blow in abdomen	Partial colectomy	Survived
13	1930	James	26	M	Duodenum	Run over by horse and cart	Evacuation	Survived
14	1933	Oppenheimer	42	F	Duodenum	Adenocarcinoma pancreas	Exploration	Died
15	1936	Larghero-Ibarz	17	M	Ileum	Needle puncture	Resection of ileum	Survived
16	1937	Glass	7 da.	M	Duodenum	Hemorrhagic diathesis	Diagnosed at autopsy	Died
17	1937	Lamy et al.	5 mo.	M	Sigmoid	Needle puncture	Diagnosed at autopsy	Died
18	1939	Smith	5 da.	F	Jejunum	? Surgical trauma	Diagnosed at autopsy	Died
19	1948	Liverud	3	M	Jejunum	Kick in abdomen	Evacuation and gastrojejunostomy	Survived
20	1951	Kratzer	70	M	Colon	Fall	Partial colectomy	Survived
21	1952	Dey	6	F	Duodenum	?	Evacuation	Survived
22	?	Schreiber	?	?	Duodenum	?	?	?
23	1952	Jones et al.	30	M	Colon	Struck by log	Partial colectomy	Survived
24	1952	Zabin	4	M	Jejunum	Fall on bicycle handlebars	Resection of jejunum	Survived
25	1953	Sachs et al.	58	M	Duodenum	?	Evacuation and gastrojejunostomy	Survived
26	1953	Stirk	9	M	Duodenum	Fall on bicycle handlebars	Evacuation	Survived
27	1954	Felson et al.	18	M	Duodenum	Blow in abdomen	Evacuation	Survived
28	1954	Felson et al.	9	F	Duodenum	Fall on abdomen	Conservative	Survived
29	1954	Felson et al.	9	M	Duodenum	Fall over a wire	Evacuation	Survived
30	1954	Felson et al.	33	M	Duodenum	?	Evacuation	Survived
31	1954	Lampert et al.	15	M	Jejunum	Kick in abdomen	Evacuation	Survived
32	1954	Magladry et al.	9	M	Duodenum	Fall from scooter	Evacuation	Survived
33	1956	Snider	11	M	Duodenum	Blow in abdomen	Exploration	Survived
34	1956	Spencer et al.	4	M	Duodenum	Blow in abdomen	Evacuation	Survived

obstruction. Of the nine patients who succumbed, only two had been explored surgically. The hemophiliac with multiple hematomas in the ileum and the patient who had a duodenal hematoma and acute pancreatitis complicating adenocarcinoma of the pancreas expired shortly after simple exploration.

It is interesting that in several patients the preoperative diagnosis was appendicitis. In a 4-year-old boy with a history of trauma, a McBurney incision was made, but the presence of bloody peritoneal fluid led to complete exploration with discovery of a large hematoma of the jejunum. In a 58-year-old man without history of trauma a retroperitoneal hematoma behind the right colon was packed at the time of appendectomy; the symptoms persisted, and ten days later exploration revealed the hematoma to obstruct the duodenum. A 9-year-old boy had an appendectomy for abdominal pain and vomiting with diffuse abdominal tenderness; vomiting continued, and several days later gastrointestinal series showed the typical deformity in the duodenum. At a second operation the hematoma was evacuated, and a history of trauma was obtained postoperatively.

PREVIOUSLY REPORTED CASES

CASE 1.[17]—A 49-year-old man with a history of digestive disturbance died of duodenal obstruction. At autopsy a "false aneurysm" was found on the posterior surface of the first portion of the duodenum. The greater curvature of the stomach measured 33 inches.

CASE 2.[3]—A 23-year-old man died of rupture of an abdominal aneurysm and at autopsy was found to have a large retroperitoneal hematoma which so compressed the duodenum that it would barely admit a finger. The stomach and duodenum were full, the jejunum empty.

CASE 3.[3]—A 33-year-old man known to have an aortic aneurysm died of esophageal obstruction a week after an episode of acute pain. At autopsy he was found to have obstruction of the lower end of the esophagus resulting from an intramural hematoma secondary to rupture of the aneurysm.

CASE 4.[28]—A 24-year-old male complained of abdominal pain, vomiting, hiccoughs, and obstipation. There were hematomas on the face and elbow in addition to an abdominal mass and tenderness. (The patient was a hemophiliac.) At surgery multiple hematomas were found in the small bowel. The patient died after surgery and at autopsy the hematomas of the bowel were found to be partially obstructing.

CASE 5.[27]—A 16-year-old man had a Bassini hernioplasty under local anesthesia and passed blood in the stool postoperatively. Intestinal obstruction developed and at operation a subserosal hematoma in the sigmoid was evacuated. The patient survived.

CASE 6.[1]—A 26-year-old man struck in the abdomen by the handlebars of a bicycle complained of vomiting, abdominal pain, and distention. At laparotomy the next day he was found to have a large subserosal hematoma of the cecum in continuity with a hematoma of the mesocolon. The history of trauma was obtained during surgery (spinal anesthesia). The ascending colon was resected and the patient survived.

CASE 7.[24]—An adult male was caught between two motor lorries and the abdomen compressed. On the third day after injury he began vomiting but had no tenderness. At operation a retroperitoneal hematoma behind the duodenum was evacuated. The patient survived.

CASE 8.[25]—A 22-year-old woman with abdominal pain and vomiting for one day was found to have abdominal tenderness and a mass in the right lower quadrant. At operation the mass was found to be a large intramural hematoma of the cecum. The appendix was

removed and the hematoma drained. The patient survived. The hematoma was attributed to injury sustained when the patient, a lady gardener, lifted a number of potted chrysanthemums the day symptoms began.

CASE 9.[14]—A 19-year-old cobbler presumably injured the right upper abdomen while making wooden shoes. When an incarcerated right inguinal hernia was reduced by the patient, a painful mass appeared in the right lower abdomen. A tender, 7 cm. mass in the right lower quadrant was found on examination. Barium enema showed no colon obstruction. At surgery seven weeks later, an old hematoma was found near the hepatic flexure. The right colon was resected and the patient survived.

CASE 10.[2]—A 6-year-old boy without a history of trauma expired shortly after admission for abdominal pain and vomiting. At autopsy he was found to have an intramural hematoma of the ileum which was causing intestinal obstruction.

CASE 11.[20]—A 7-year-old boy complained of abdominal pain and vomiting and was found to have epigastric distention and an epigastric mass the size of a lemon. At operation a 5 × 10 cm. hematoma of the jejunum was evacuated. The patient survived.

CASE 12.[6]—A 24-year-old sawmill worker was repeatedly struck in the abdomen during the course of his work. He complained of constipation of three years' duration and was found to have a mass in the right abdomen. The barium enema showed a filling defect in the ascending colon, and subsequently a partial colectomy was done for an organized intramural hematoma. The patient survived.

CASE 13.[8]—A 26-year-old man was run over by a horse and cart and six days later complained of abdominal pain and vomiting. On the eleventh day after injury a mass was palpated to the right of the umbilicus. At surgery the next day a hematoma the size of a tennis ball behind the third portion of the duodenum was evacuated. The patient survived.

CASE 14.[18]—A 42-year-old woman was admitted with vomiting and abdominal distention and tenderness. At operation the stomach was emptied of 3,500 c.c. of fluid; acute pancreatitis with fat necrosis was found. The patient died immediately after surgery and at autopsy was found to have acute pancreatitis with fat necrosis, a hematoma on the posterior wall of the second portion of the duodenum, and adenocarcinoma of the pancreas.

CASE 15.[13]—A 17-year-old boy was operated on for an incarcerated inguinal hernia under local anesthesia. In injecting anesthesia into the constricting ring at the neck of the sac, the incarcerated loop of ileum was inadvertently punctured. The resulting small hematoma was controlled by pressure. On the third day after surgery, continued vomiting and distention led to a diagnosis of intestinal obstruction due to increase in the size of the hematoma. At operation the diagnosis was confirmed. The serosa over the hematoma had ruptured, resulting in a large intraperitoneal hemorrhage. The involved ileum was resected and the patient survived.

CASE 16.[7]—A 1-day-old Negro male infant vomited blood and coffee-ground material. He continued to bleed, became icteric, and exhibited a generalized hemorrhagic diathesis. After death on the seventh day, autopsy revealed a submucosal hematoma obstructing the duodenum and the common duct.

CASE 17.[12]—A 5-month-old male infant who survived severe bilateral pneumonia and erysipelas developed diarrhea with vomiting and was treated with subcutaneous and intraperitoneal fluids. The patient died of intestinal obstruction and at autopsy was found to have a large submucosal hematoma of the rectosigmoid, supposedly produced by needle puncture.

CASE 18.[22]—A white female infant with atresia of the esophagus and tracheoesophageal fistula (Type C) had a gastrostomy on the third day of life. The infant vomited and finally

aspirated and died on the fifth day. Autopsy findings included a small hematoma around the gastrostomy and a large submucosal hematoma obstructing the proximal jejunum.

CASE 19.[15]—A 3-year-old boy was kicked in the abdomen and vomited for three days. Gastrointestinal series revealed a typical intramural hematoma in the upper jejunum. At operation the hematoma was evacuated and a gastrojejunostomy done because of questionable viability of the involved bowel. The history of trauma was obtained postoperatively. The patient survived.

CASE 20.[10]—A 70-year-old man fell six months before admission and complained of diarrhea of three months' duration. Barium enema showed a constricting deformity of the ascending colon. The patient survived a right hemicolectomy for an intramural hematoma of the colon.

CASE 21.[4]—A 6-year-old girl had severe abdominal pain for two days, began vomiting, and was found to have a nontender mass to the right of the umbilicus. At operation a hematoma was found on the anterior wall of the first and second portions of the duodenum. The hematoma was evacuated and the child survived.

CASE 22.[21]—A case of duodenal obstruction due to intramural hematoma similar to Case 21 was not described in detail.

CASE 23.[9]—A 30-year-old man complained of abdominal pain and constipation two months after being struck in the abdomen by a log. There was progressive abdominal distention but no mass or tenderness. Barium enema showed a constricting lesion in the descending colon. The lesion was resected and proved to be an organized intramural hematoma. The patient survived.

CASE 24.[29]—A 4-year-old boy fell on the handlebars of a bicycle five days prior to admission and three days later complained of abdominal pain and vomiting. There was vague tenderness in the lower abdomen. At appendectomy bloody peritoneal fluid was found, and a 14 inch segment of jejunum, 2¾ inches in diameter, was resected. The hematoma was found to be submucosal. The patient survived.

CASE 25.[19]—A 58-year-old man had abdominal pain and vomiting for two days and was admitted with abdominal distention and tenderness. An appendectomy was done and a retroperitoneal hematoma behind the right colon packed. A few days later, x-rays revealed duodenal obstruction and a mass in the right abdomen. At operation, ten days after appendectomy, a large retroperitoneal hematoma invading the leaves of the mesentery and obstructing the duodenum was partially evacuated. A gastrojejunostomy was done, and the patient survived.

CASE 26.[26]—A 9-year-old boy fell across the handlebars of his bicycle five days before admission and three days later began to complain of abdominal pain and vomiting. There was little abdominal tenderness and a mass in the midline above the umbilicus. At operation a hematoma involving the second, third, and fourth portions of the duodenum and the first 4 cm. of the jejunum was evacuated. The patient survived.

CASE 27.[5]—An 18-year-old man who was struck in the abdomen while playing football complained of abdominal pain and vomiting. Gastrointestinal series showed the typical deformity. At operation a 20 cm. subserosal hematoma was found in the distal duodenum and proximal jejunum, communicating with a large retroperitoneal hematoma. The hematoma was evacuated and the patient survived.

CASE 28.[5]—A 9-year-old girl fell, injuring the abdomen, and complained of abdominal pain and vomiting. There was moderate abdominal tenderness. Gastrointestinal series showed the typical deformity in the duodenum just proximal to the ligament of Treitz. The child survived with conservative treatment.

CASE 29.[5]—A 9-year-old boy fell over a wire, complained of abdominal pain and vomiting, and was found to have diffuse abdominal tenderness. Appendectomy was done, but symptoms persisted. Gastrointestinal series showed the typical deformity. At operation ten days after appendectomy a hematoma on the posterior surface of the duodenum was evacuated. The child survived, and the history of trauma was obtained postoperatively.

CASE 30.[5]—A 33-year-old man complained of hiccoughs and vomiting for two days. There was no history of trauma. Gastrointestinal series showed the typical deformity. At operation a large subserosal hematoma of the transverse duodenum was evacuated. The patient survived.

CASE 31.[11]—A 15-year-old boy was kicked in the abdomen while playing football and complained of vomiting and upper abdominal pain and tenderness for two days. Gastrointestinal series showed obstruction of the proximal jejunum, and a mass was palpated to the left of the midline. At operation a 3 by 5 inch hematoma of the jejunum was evacuated. The patient survived.

CASE 32.[16]—A 9-year-old boy fell from a scooter two days before admission and complained of abdominal pain and vomiting. X-rays revealed duodenal obstruction. At operation, cardiac arrest was treated by cardiac massage, then exploratory laparotomy was done. A submucosal hematoma of the duodenum was explored by gastrotomy and duodenotomy and then evacuated. The patient survived.

CASE 33.[23]—An 11-year-old boy was struck in the abdomen while playing football and the following day complained of epigastric pain and vomiting. A firm tender mass was palpated in the right upper quadrant of the abdomen. Gastrointestinal series showed the typical deformity of the third portion of the duodenum. At surgery 13 days after injury the hematoma was found but was not evacuated. The patient was fed for two weeks through a tube passed into the jejunum, following which he made an uneventful recovery.

CASE REPORT

D. G., a 4-year-old white boy, was admitted Dec. 14, 1955, with the following history: he had three loose, watery bowel movements on the evening of December 10 and another the following morning when he also had anorexia. About noon that day he ate three kumquats* and several hours later vomited some of the peel and pulp of the fruit and began to complain of epigastric pain. He had a normal bowel movement the next day but continued to vomit. On December 13 the patient was admitted to the local hospital and given intravenous fluids; the following morning a gastrointestinal series showed complete pyloric obstruction, and therefore the patient was transferred to the Charity Hospital.

There was no hematemesis or melena, and the vomitus was said to contain no bile. There was no past history of vomiting.

The only significant findings on physical examination were mild elevation of temperature and slight guarding in the right upper quadrant of the abdomen. The white count was 10,900 with 86 per cent polys. X-rays revealed considerable gastric retention of the barium administered 12 hours previously, but some contrast material was visible in the small bowel.

Nasogastric suction and intravenous fluids were started and the child was given sedation. When the abdomen was re-examined, the guarding had disappeared, but there was a questionable mass beneath the upper portion of the right rectus muscle.

The day after admission, December 15, the abdomen was soft and flat, peristalsis was normal, and there was no tenderness or guarding. No mass could be outlined but there remained a suggestion of fullness in the right epigastrium. Repeat films of the abdomen showed the stomach to be empty (via the Levin tube) but the duodenum was irregularly outlined by barium (Fig. 1).

*A small citrus fruit of which both peel and pulp are often eaten.

Fig. 1.—X-ray 24 hours after the administration of barium shows the stomach to be almost empty (via the Levin tube). The first portion of the duodenum is well outlined, but the second portion shows the irregular filling defect laterally (arrow) in the area of the intramural hematoma. Some contrast material is seen in the distal small bowel and in the ascending colon.

Fig. 2.—The second gastrointestinal series revealed the obstruction in the first portion of the duodenum. The rounded filling defect (arrow) at the site of the obstruction suggests extrinsic pressure but was thought perhaps to represent a kumquat impacted above a partial congenital obstruction.

The following afternoon, December 16, the child complained of epigastric discomfort, vomited, and was found to have epigastric distention and visible waves of peristalsis over the upper abdomen. The Levin tube was reinserted, and the following morning the gastrointestinal series was repeated. The level of obstruction was found to be in the first portion of the duodenum just distal to the pylorus (Fig. 2). The patient was scheduled for exploratory laparotomy with a preoperative diagnosis of duodenal obstruction of unknown etiology. It was thought unlikely that the kumquats alone were causing the obstruction, but it was considered possible that the fruit may have become impacted above a previously asymptomatic annular pancreas or congenital stenosis of the duodenum.

On December 20, exploration of the upper abdomen revealed a dark-blue retroperitoneal mass covering the first three portions of the duodenum (Fig. 3). Extending laterally from the duodenum was a small amount of retroperitoneal discoloration resembling old hematoma. By insertion of a finger into the foramen of Winslow it was possible to determine that the mass was primarily periduodenal, not diffusely retroperitoneal, and that it was fairly freely movable.

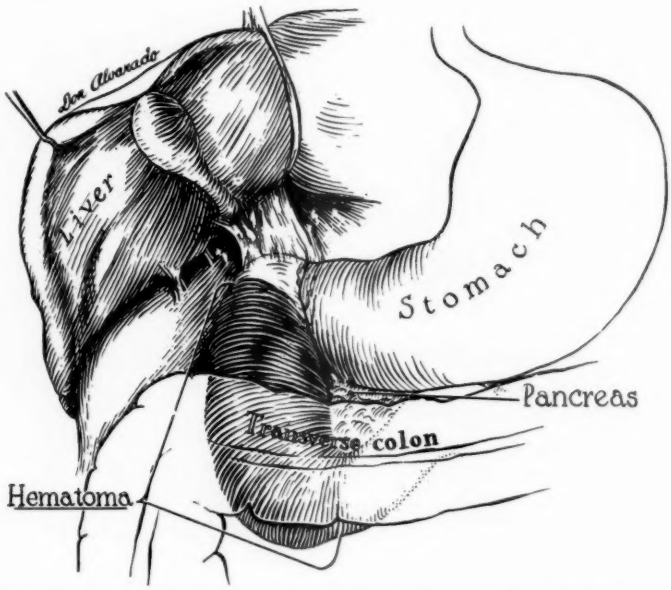

Fig. 3.—The artist's conception of the operative findings shows the location of the intramural hematoma. The duodenal cap was normal, but the remainder of the duodenum was obscured by a large dark mass extending behind the transverse mesocolon.

The peritoneum was incised lateral to the duodenum and a large amount of old blood, both liquid and clotted, was evacuated. There was no evidence of perforation into the lumen of the duodenum. The common bile duct was not identified, but it was presumed to course through the mass. Drains inserted through the incision in the posterior peritoneum were brought out through a lateral stab wound. The patient tolerated the procedure without difficulty.

After operation the patient's mother stated that the day the child began vomiting, December 11, he had fallen on a stick, striking the right upper abdomen. There was no abrasion or ecchymosis to indicate the site of the injury. Obviously the diarrhea and the kumquats were unimportant, but it is doubtful that the correct diagnosis would have been made preoperatively even had the history of trauma been known.

The postoperative course was completely uneventful. There was never any significant drainage from the stab wound or from the incision. On Christmas Day, four days after

operation, the child ate and retained large quantities of turkey and dressing. Gastrointestinal series nine days postoperatively showed no abnormality, and the child was discharged from the hospital that afternoon. He has been followed in the outpatient clinic since discharge and had remained quite well.

DISCUSSION

Intramural hematoma is a rare complication of blunt trauma to the abdomen. As with other nonpenetrating injuries, it occurs most often in the relatively fixed portions of the gastrointestinal tract. Only mild symptoms of pain and tenderness are noted immediately after the injury; then, as the hematoma increases in size and dissects circumferentially and longitudinally in the bowel wall, intestinal obstruction becomes apparent. The interval between injury and onset of symptoms of obstruction may vary from a few hours to several days.

A history of trauma may not always be obtained in these lesions, but they often will be accounted for by some associated condition such as retroperitoneal hemorrhage or bleeding disorder.

Generally the physical examination reveals little or no abdominal tenderness, and only rarely will a mass be palpable. The diagnosis may be made by gastrointestinal series; in the typical case, the hematoma bulges into the lumen, creating a filling defect, and the folds of mucosa are made prominent by edema and hemorrhage, producing the coil-spring appearance.

In most cases simple evacuation of the hematoma will suffice, but if there is an extensive lesion an enteroenterostomy may be indicated. In the case of an organized intramural hematoma of the colon, partial colectomy may be necessary to exclude the possibility of malignancy. When there is questionable viability, the involved intestine should be resected if possible.

SUMMARY

A case of intramural hematoma of the duodenum is reported. This makes a total of 34 published cases of such lesions producing intestinal obstruction. The literature has been reviewed and the previously published cases summarized. Many of the lesions were traumatic, and simple evacuation or resection of the hematoma resulted in survival in every case so treated.

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A METHOD OF OPERATING ON FETAL DOGS IN UTERO

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INTRODUCTION

THE method to be described is one of operating on fetal dogs and then leaving the operated pup to develop further in utero and be born spontaneously at term. The technique was developed from experiments which were designed to prove that at least some cases of congenital ileal atresia are due to an interference with the ileal blood supply in utero (Louw and Barnard, 1955).[1]

Fetuses were subjected to surgery 40 years ago by Mayer (1915),[2] and since then this experimental procedure has been carried out successfully by various workers. The pertinent literature has been well reviewed by Wells (1950).[3] Most of these reported experiments were performed an small animals such as rats, cats, and rabbits. Here the size of the fetus makes the procedure technically more difficult. Furthermore, the operated fetus usually has been left in an extrauterine location in the peritoneal cavity with an intact placental blood supply to develop further.

As far as can be determined, the experiment about to be described is the first of its kind reported and it is the first time that the origin of congenital malformation of the gastrointestinal tract has been studied by directly interfering with the blood supply of the fetal bowel. The continued success we have had since our first report makes us hopeful that these experiments will open a new field to research in congenital abnormalities, tissue and organ transplantation, and the study of fetal physiology and metabolism.

MATERIALS AND METHODS

Mongrel bitches, of average size, 45 to 55 days pregnant were utilized.

The pregnant animal is anesthetized with intravenous pentobarbital (1 grain per 5 pounds of body weight). The operative field is prepared by shaving the right half of the abdominal wall.

An oblique incision is made from below the tip of the right thirteenth rib to the medial border of the right rectus abdominus muscle midway between the umbilicus and pubis. The three abdominal muscles are split parallel to their fibers and the peritoneum is incised.

The gravid uterus is carefully palpated and the fetus situated in the middle of the right horn is located by palpation of the gravid uterus. This portion

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of the uterus is brought into view in the abdominal wound. At no stage of the operation is any portion of the gravid uterus delivered from the peritoneal cavity. This precaution prevents any danger to occlusion of the uterine vessels.

The placental sites can be identified by the course of the uterine vessels and by palpation of the uterine wall. The uterus should be sectioned some distance from the placental site in order to avoid dividing large vessels and to minimize the danger of placental separation.

Before incising the uterus, however, the exact position of the fetus with respect to the uterus is determined by palpation. When the fetal parts have been so identified, the fetus is rotated so that the area of the fetus to be operated upon comes to lie adjacent to the part of the uterine wall to be incised. The puppy is then held in this position by an assistant, and with a knife the uterus is opened for about ¾ to 1 inch. The bleeding from the uterine wall should be minimal.

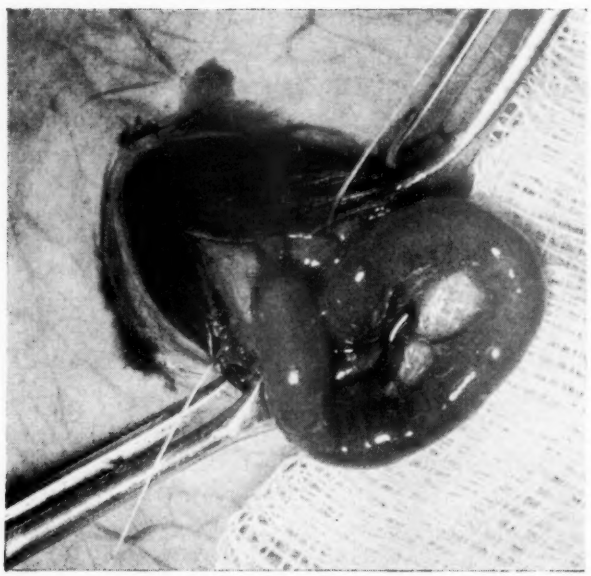

Fig. 1.—Photograph showing a loop of fetal ilium delivered. Note that only a small segment of the uterus is outside the peritoneal cavity of the mother dog and that traction on the skin of the puppy is maintained to prevent further escape of amniotic fluid.

Once the uterine cavity is entered, the skin of the fetus, which is very mobile, is immediately picked up in a pair of dissection forceps and drawn into the uterine wound. The fetus will now act as a stopper and prevent the escape of amniotic fluid. If the position of the puppy was correctly identified, the area presenting in the uterine wound should be the operative field in the fetus. It is important not to allow the fetus to escape from the uterus as the uterus will then immediately contract and replacement of the fetus then becomes impossible. In some of our experiments the cord has prolapsed. If this should happen, it must be gently replaced. The fetus is now in a position where it can be operated upon without interfering with the placental blood supply. If any difficulty is experienced in determining the exact position of the puppy, a

limb can be delivered after the uterine cavity has been entered. By studying the limb, whether front or back, the position of the fetus should become apparent.

In our experiments concerning the etiology of congenital ileal atresia, the peritoneal cavity of the fetus was opened by incising the skin with an iridectomy scissors for ½ to ¾ inch. The abdominal muscles were grasped and all layers of the fetal abdominal wall were incised simultaneously. Once the fetal peritoneal cavity was entered, a loop of ileum was delivered and one or more of the ileal vessels was ligated with nonabsorbable material (Fig. 1). The infarcted ileum was replaced and the abdominal wound was closed by suturing the muscles and peritoneum together with a few interrupted plain catgut stitches (3-0) and stitching the skin with two or three interrupted (4-0) silk stitches. The uterine wound was closed by means of a running catgut 3-0 stitch. No attempt was made to suture the membranes.

The abdominal wound of the mother was closed in layers.

In some cases, a fetus in the left horn was selected and operated upon in a similar fashion.

RESULTS

Before the method described was evolved, our results were invariably bad. The mother either aborted soon after operation, or the fetus operated on was born at term in a macerated state. We believe that this is due to two main reasons: namely, (1) interference with the main uterine vessels and (2) interference with the placental circulation of the puppy operated upon. The first complication can be prevented by not delivering the gravid uterus from the abdominal cavity of the mother, thus preventing any drag on or compression of the main uterine vessels. The second complication can be prevented by making the uterine incision away from the placental site and by the operative technique described, which prevents the escape of large quantities of amniotic fluid and which leaves the fetus inside the uterine cavity during the experimental procedure. Since the evolution of the new technique, 80 per cent of the puppies operated upon in utero were born alive.

SUMMARY

A method of operating on a fetal dog 14 days before term and then leaving it in utero to develop fully is described. There seems to be no reason why operations could not be done at an early stage of gestation with expectation of success.

It is proposed that this technique may serve as a useful tool in investigating other congenital malformations, fetal metabolism, and physiology.

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ACUTE FOCAL CEREBRAL ISCHEMIA

Role of Interruption of Sympathetic Impulses to the Cerebral Vessels in Its Prevention and Treatment

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MUCH disagreement persists concerning appropriate measures for prevention of acute attacks of focal cerebral ischemia and progression to ischemic or hemorrhagic infarction of the involved area of the brain. There is also no agreement regarding the most common precipitating factor of such attacks and the mechanism of progression, in some instances, to hemorrhagic infarction of the affected area of the brain.

The pathologic condition primarily responsible for development of a syndrome compatible with the diagnosis of acute focal cerebral ischemia may not be evident, but those that usually receive primary consideration are occlusion of the common or internal carotid artery by a thrombus or embolus, cerebral arterial thrombosis, cerebral embolism, or cerebral vasospasm. Frank intracerebral hemorrhage may produce neurologic deficits equally as acute as those caused by cerebral embolism, but the former usually is accompanied by more pronounced alteration of the vital signs. If interruption of cerebral blood flow is secondary to an embolus or thrombus, there undoubtedly is an added factor of vasospasm, which further interferes with development of adequate cerebral circulation by anastomotic channels.

The probable importance of vasospasm in such cases was suggested experimentally with cerebral emboli by Villaret and Cachera.[45] They noted some degree of spasm in all ipsilateral cerebral vessels and often lesser constriction in the contralateral vessels. Vasospasm and edema distal to thrombotic occlusion of a peripheral artery of an extremity are often observed clinically. Westphal and Bär[47] theorized that focal spasm of a cerebral vessel might be sufficiently severe and prolonged to produce changes secondary to anoxia in the walls of the involved arterial tree, and with resumption of more normal blood flow through the affected vessels, blood could be forced into the tissues of the brain. Schwartz[41] concurred essentially with this theory, adding that by the confluence of such petechial hemorrhages a large intracerebral hematoma might form.

Aring[6] also postulated that a cerebrovascular accident might be primarily a vasomotor phenomenon with a period of undue vasoconstriction followed by reactive vasodilatation. He suggested that oxygenation of the tissues must

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continue to be grossly inadequate during the period of vasoparalysis and that the relatively thin walls of the capillaries and venules are the most vulnerable sites for extravasation of blood. Stern[43] concluded that necrotic lesions of the brain may occur without associated visible obstruction of the arteries. He reasoned that since the shape and distribution of such lesions suggest their arterial origin, they may result from a vasomotor disturbance. Hicks and Black[20] found no evidence of mechanical vascular obstruction in 11 of 23 cases in which the fatal lesion, in each case, was predominantly cerebral infarction. A drop in systemic blood pressure was a possible contributing factor in only 2 cases. Later, Hicks and Warren[21] reported no mechanical obstruction of the cerebral vessels in 60 of 100 cases of fatal cerebral infarction. In some instances, at autopsy, several smaller branches of the arterial tree may be found to be occluded by thrombi rather than the major artery as was suggested clinically. This might be interpreted as representing a reaction of vasospasm that was sufficiently prolonged for thrombi to form in several of the branches of the involved artery. On the other hand, multiple areas of obstruction in an arterial tree might actually represent emboli from a reaction about an atheromatous lesion that has developed on the endothelial surface of the parent artery. Zak and Elias,[48] for example, showed that atheromatous lesions of the aorta or of the common or internal carotid arteries can be the source of cerebral emboli.

Fisher and Adams[13] suggested that hemorrhagic infarction of the brain indicates occurrence of embolism. They theorized that an embolus may arise from an atheromatous reaction at the level of the carotid siphon and, after causing infarction of tissues distal to the point of original obstruction, may move farther peripherally, owing to fragmentation of the embolus, relaxation of local vascular spasm, or both. It was postulated that this caused occlusion of multiple smaller arterial vessels more distally located, followed by exposure of tissues, between the first and second points of arterial obstruction, to the full force of the systemic arterial pressure, which in turn results in hemorrhages from the damaged vessel walls between these points and irreparable hemorrhagic infarction of the brain. Others pointed out that a thrombus forming opposite an atheromatous lesion, for example, of the carotid siphon, may be the source of one or more emboli with a resultant cerebrovascular accident. It has been suggested[33] that such emboli may completely disintegrate and consequently cause no permanent obstruction. Thus, no obstruction might be demonstrated angiographically or by direct examination at autopsy. Still others[12] expressed the opinion that emboli may arise from a thrombotic reaction about atheromatous lesions that have involved the intima of the large proximal arteries of the brain. It therefore is evident that careful correlation of the history, clinical picture, and laboratory data is essential for satisfactory evaluation of any cerebrovascular accident.

There appears to be considerable evidence that cerebral vasospasm, in addition to complicating the syndromes of carotid or cerebral arterial thrombosis and cerebral embolism, can cause transient periods of focal cerebral

ischemia with short periods of neurologic deficits. Also, vasospasm alone apparently can cause ischemic or hemorrhagic infarction of the brain with subsequent persistent neurologic deficits. If it is agreed that spasm of a portion of the cerebral arterial tree may be a fundamental factor in the appearance of the neurologic deficits constituting a stroke, the question arises as to why one relatively small portion of the cerebral arterial system should react in such a manner. The answer is not obvious but it might be pointed out that evidence of local dysfunction of the autonomic nervous system is frequently seen. Localized areas of vasomotor instability in the skin, particularly of the peripheral portions of the extremities, or in the gastric mucosa are common examples of such focal dysfunction of smooth muscle. Similarly, migraine apparently is the result of localized vasoconstriction followed by pronounced vasodilatation. The primary precipitating factor may be obvious, such as undue excitement, but the cause of a particularly severe reaction in some localized area may be equally unapparent. Paterson[36] believed that intimal hemorrhages from ruptured capillaries in sclerotic cerebral arteries may cause focal vasospasm. It has been theorized that the factors responsible for rupture of the intimal capillaries are high intracapillary pressure, progressive atheromatous degeneration of the tissues supporting the capillaries, and undue capillary fragility from other causes.

Although some have minimized the role of vasospasm in the development of acute focal cerebral ischemia, even insisting that it is physically impossible for the cerebral vessels to constrict sufficiently to do so "because of the poorly developed media," almost all workers implicate vasospasm to some extent in theorizing regarding the probable mechanism of cerebrovascular accidents. It has been denied that there are vasomotor nerves to the cerebral vessels.[7] However, Stöhr[44] and others[9, 38] presented evidence of such nerves. It has, of course, been repeatedly demonstrated at operation and angiographically that the cerebral vessels can constrict to a sufficient degree almost to obliterate the lumen of the involved artery. Therefore, multiple factors and many contradictory reports must be considered in the evaluation of measures to prevent undue vasospasm or to cause local vasodilatation in the prevention of attacks of acute focal cerebral ischemia. Similarly, determination of the best plan of treatment of a cerebrovascular accident, consisting of persistence of the neurologic deficit that began as an acute bout of focal cerebral ischemia, requires consideration of many contradictory reports.

The anastomotic relations of the major intracranial arteries at the circle of Willis, of the major cerebral arteries in the pia, and of the intrinsic cerebral arterioles constitute a great potential for the development of adequate collateral circulation. In addition, anastomotic connections between the external carotid arterial system and the ophthalmic artery may represent minor collateral circulation for the brain. The question regarding the possible greater exploitation of these collateral channels, with the appearance of evidence of focal cerebral ischemia, by interruption of the sympathetic impulses to the cerebral vessels of the involved hemisphere, has continued, however, to be

controversial. Favorable reports[3, 4, 16-19, 23, 27-30, 34, 35, 37, 39, 40, 42, 46] greatly outnumber unfavorable reports[1, 2, 5, 10, 11, 31, 32] concerning use of sympathetic blocks in the treatment of such patients. Those rendering favorable reports have, in essence, observed that many patients of this group demonstrate some improvement of motor function within a few minutes of the development of a satisfactory Horner's syndrome. The most logical conclusion is that the sympathetic block has, to some extent, lessened the degree of cerebral ischemia allowing for improved motor function in the muscles supplied by the affected motor cortex. This improvement may or may not persist, but its occurrence in this temporal relation to the sympathetic block is rather convincing evidence of the potential efficacy of the treatment.

Adverse criticism regarding interruption of the sympathetic impulses to the cerebral vessels for acute focal cerebral ischemia has been expressed by several authors. Denny-Brown[11] criticized the "indiscriminate" use of stellate block in the treatment of cerebrovascular accidents, citing the case of a 68-year-old hypertensive woman who had failed to demonstrate any improvement of left hemiplegia although the neurologic resident blocked the right stellate ganglion with 6 c.c. of a 1 per cent solution of procaine 30 minutes after onset of the paralysis. At autopsy, 4 weeks later, the right internal carotid artery was completely occluded and the basilar artery almost completely occluded by atheromas and superimposed thrombosis. He concluded that this appeared to be a test case for the value of the procedure. Alvarez[2] theorized that a "little stroke" may be caused by a "little spasm in an artery" and concluded that the clinical evidence of the stroke may disappear because "the collateral circulation may have opened up" but stated that he had "no faith" in sympathetic blocks in the treatment of such cases. He did not mention having used them.

Millikan and associates,[31] who evaluated "stellate ganglion block for acute focal cerebral infarcts," failed to find evidence that they were beneficial. In fact, they concluded that sympathetic block had actually been detrimental to some of their patients. Gilbert and de Takats[16] had previously suggested that sympathetic blocks should not be performed on patients in whom it was thought that an intracerebral hemorrhage constituted the cerebrovascular accident. In sharp contrast to the relatively few unfavorable reports regarding use of sympathetic blocks in improving cerebral circulation locally, following clinical application of the treatment, is the large number of reports now available confirming the favorable reactions observed by Leriche and Fontaine,[28] who first gave this type of treatment adequate clinical trial.

For the past several years, we[23] have interrupted the sympathetic impulses to the vessels of the involved cerebral hemisphere by infiltration of procaine about the cervical sympathetic trunk or by surgical section of the cervical sympathetic pathways in a small group of patients with clinical manifestations of acute focal cerebral ischemia that did not subside shortly or in persons who had recurring attacks with alarming frequency. Because of the continued controversy concerning appropriate preventive measures and therapy

for such attacks and because of the difficulties encountered in attempting to reach a more definite decision to the problem by experimentation, we believe that at present the greatest contribution to its solution is the objective evaluation of the response of patients with acute focal cerebral ischemia to this procedure. We wish, therefore, to present 4 cases of acute focal cerebral ischemia, each of which represented special problems in cerebral circulation, and to describe their reactions to interruption of the sympathetic impulses to the cerebral vessels.

CASE REPORTS

CASE 1.—M. F. H., a right-handed, 49-year-old farmer, was admitted to the Ochsner Clinic, Oct. 25, 1951, complaining of attacks of weakness of the right arm and leg and difficulty in speaking. Six weeks earlier, upon becoming excited, he suddenly noted extreme weakness of the right extremities and inability to express his thoughts normally. After resting quietly for 15 or 20 minutes, he was able to resume his usual activities as the symptoms subsided. Forty-eight hours later a similar episode occurred. The patient consulted his local physician, but by the time he was examined approximately one hour later, the symptoms had again subsided. Examination by his physician disclosed nothing of significance except that the systolic blood pressure was 140 mm. Hg and the diastolic pressure was 100 mm. Hg. Similar attacks occurred almost daily during the subsequent 6 weeks and were more likely to occur when he became excited or unduly fatigued.

The patient came to the Clinic "because he was afraid he was going to have a stroke." Examination revealed nothing of significance except mild spasm of the vessels of the optic fundi and a blood pressure of 142 mm. Hg systolic and 100 mm. Hg diastolic. The peripheral blood studies and the blood chemistry determinations revealed no variations from normal. Intravenous pyelography, electroencephalography, electrocardiography, and roentgenography of the chest and skull revealed no evidence of pathologic alterations. The pineal gland was in the normal position. The cerebrospinal fluid pressure was normal and the cerebrospinal fluid survey revealed no evidence of disease.

The consensus was that the patient was having attacks of circulatory insufficiency of the left internal carotid system. It seemed possible that an important part of the pathologic picture was bouts of undue spasm of the middle cerebral artery because of their association with undue excitement. Therefore, left superior cervical ganglionectomy was recommended. The patient, however, decided to postpone the operation for 2 weeks in order to complete some work. He returned one week later with severe right hemiparesis and moderately severe expressive aphasia. On the preceding day he had had 7 attacks of weakness of the right extremities and difficulty in speaking. The last attack had not cleared after approximately 18 hours had elapsed. Left cervical sympathetic block was followed, within 3 or 4 minutes, by pronounced improvement in the strength of the right extremities and in the ability to speak. On the following day, left superior cervical ganglionectomy was performed, a local anesthetic being used. This was well tolerated and the symptoms completely subsided within 48 hours. Symptoms had not recurred by the time of discharge on the fifth postoperative day. Three months after operation, there still had been no recurrence of neurologic deficits; therefore the patient was allowed to resume all former activities. At this writing, more than 4 years postoperatively, there still has been no recurrence of the syndrome.

Comment: The factor of spasm of the cerebral arteries seems well demonstrated in this case. Although numerous transient attacks of right hemiparesis and partial expressive aphasia may be the clinical manifestations of several neurologic disorders, the most logical conclusion in this instance is that they represented either only attacks of severe spasm of the left middle cerebral artery or occlusion of the internal carotid artery supplemented by undue spasm of the middle cerebral artery. The gratifying response to excision of

the left superior cervical sympathetic ganglion supports both contentions. That there was a close functional relationship between the periods of neurologic deficits and the sympathetic nervous system seems obvious when one considers the fact that such attacks occurred one or more times almost daily for 7 weeks prior to sympathectomy and none has occurred in the 4 years since. This case also appears to demonstrate that spasm of the cerebral vessels can be sufficiently severe and prolonged to produce permanent damage to the brain, although in this instance complete recovery followed sympathectomy.

CASE 2.—V. B., a 37-year-old oil field worker, was admitted to Ochsner Foundation Hospital, May 2, 1953, complaining of severe headache, pain in the back of the neck, inability to open the left eye and double vision when it was held open. About 3 weeks earlier the patient had begun to experience severe pain behind the left eye. The discomfort, which was present at all times following the onset, was punctuated by severe exacerbations of pain. After one week, the pain forced him to stop working. One week prior to admission, as he arose from a chair, the retro-orbital pain suddenly became much worse. This was quickly followed by pain and stiffness of the neck. Shortly thereafter, he noted double vision and difficulty in opening the left eye. This became progressively worse during the subsequent 24 hours, until he could not open the affected eye. The severe pain persisted. Two days before admission he had another attack of extremely severe pain in the head followed shortly by severe pain and stiffness of the neck.

On admission, the patient was slightly confused but presented no other neurologic deficits than paralysis of the muscles supplied by the left oculomotor nerve. The blood pressure was 120 mm. Hg systolic and 82 mm. Hg diastolic, and the pulse rate was 80 beats per minute. Lumbar spinal puncture revealed grossly bloody cerebrospinal fluid under a pressure of 320 mm. of the fluid. Percutaneous angiography confirmed the diagnosis and localization of the aneurysm.

Two hours after admission, a carotid clamp was placed on the left internal carotid artery just distal to its origin. The clamp was closed sufficiently to reduce the diameter of the lumen of the vessel approximately one-half. It was not closed completely at operation since a general anesthetic had been given because of the patient's extreme restlessness and inability to cooperate. The left oculomotor nerve dysfunction remained unchanged but the confusion cleared. The left retro-orbital pain was less intense but continued to be severe. Twelve hours later the artery was completely occluded. This further relieved the pain and did not alter the neurologic status. On the fifth day after admission, the internal carotid artery was occluded intracranially by application of a silver clip immediately proximal to its bifurcation. Upon awakening from the general anesthetic, the patient had no retro-orbital pain. However, almost 36 hours postoperatively, a moderate degree of right hemiparesis and expressive aphasia developed. Within 4 minutes following cervical sympathetic block on the left, considerable improvement was noted in the aphasia but the hemiparesis only partially resolved. Since this degree of improvement was not maintained, the sympathetic block was repeated on the following day with complete disappearance of the hemiparesis and aphasia. However, on the fifth day following occlusion of the carotid artery intracranially, when the patient was allowed to get out of bed, paralysis of the right upper extremity and a central type of paralysis of the right side of the face developed. There was pronounced paresis of the muscles of the right lower extremity. Expressive aphasia was also present. The degree of consciousness was not altered and the patient continued to be free of the retro-orbital pain. Cervical sympathetic block was performed at once and at 12- to 24-hour intervals for one week. Moderate improvement followed each sympathetic block. The degree of improvement apparent within a few minutes after each block never persisted in its entirety but was never completely lost. The patient was discharged on the fourteenth day after the second operation with only a slight degree of right hemiparesis and aphasia. He continues to be well after 30 months have elapsed postoperatively.

Comment: This case is of particular interest physiologically. Following obstruction of the left internal carotid artery as a result of trapping the aneurysm arising from it,

the major source of blood for the contralateral cerebral motor cortex, of course, was the opposite internal carotid artery. Thus, one might expect that the cerebral circulation on the side of the occluded internal carotid artery would be more defective in the area of the middle cerebral than in the distribution of the anterior cerebral artery. This was demonstrated clinically. The blood supply to the area of the cerebrum in question was adequate until the patient spent some time in an erect position upon being out of bed for the first time postoperatively. The appearance of evidence of cerebral ischemia at this time would suggest that the circulatory system had barely compensated for the arterial occlusion, and the slightly added requirement placed upon it with the patient erect was just enough to prevent maintenance of adequate oxygenation of the cortex in the distribution of the middle cerebral artery, that is in the area most distal from the major source of blood. This was manifest clinically by paralysis of the muscles of the right side of the face and the right upper extremity, expressive aphasia, and paresis of the right lower extremity. Of special interest is the fact that the clinical evidence of cerebral ischemia appeared long enough after the artery had been occluded so that any spasm that might normally occur with occlusion of an artery, either surgically or by an embolus or thrombus, should not have been a factor in this instance. It is especially noteworthy, therefore, that cervical sympathetic block was obviously effective in improving the cerebral circulation. This case seems to indicate that sympathetic block may do more than merely cause some relaxation of "abnormally constricted" vessels. The response of this patient to sympathetic blocks was also particularly gratifying because it demonstrates the fact that restoration of the patency of the internal carotid artery following its occlusion in the treatment of an aneurysm may be avoided by use of sympathetic blocks and may in this way greatly alter the prognosis relative to the aneurysm.

CASE 3.—Mrs. E. M., a right-handed, 77-year-old woman, was admitted to Ochsner Foundation Hospital with pitting edema of the legs and feet and moderate dyspnea. Twelve hours earlier she had awakened to find that she was unable to arise from her bed because of paralysis of the left extremities. The muscles of the left side of the face, except for the frontalis and the orbicularis oculi, were paretic. She was rational and explained that the paralysis had apparently developed while she slept. She had experienced no headache prior to going to sleep or on awakening. However, for several days she had noted transient attacks of numbness and tingling and a sensation of heaviness of the left extremities, which seemed to be relieved by lying down. In addition to the paralysis, there was considerable diminution in the normal acuity of exteroception and proprioception over the left half of the body and left extremities. There was no aphasia.

The patient was admitted to the medical service. She was immediately given caffeine sodium benzoate and a mercurial diuretic and placed under an oxygen tent. These measures made her more comfortable but the paralysis persisted unchanged for an additional 24 hours. Right cervical sympathetic block was then performed; it produced a satisfactory Horner's syndrome. After 4 minutes the tone of the muscles of the involved extremities had improved and there were weak voluntary movements of the flexor and adductor muscles of the arm. Slight voluntary movement of the muscles of the left thigh and leg was noted. This degree of improvement persisted for approximately 2 hours. At the time of the second sympathetic block 12 hours later, the left upper extremity was again paralyzed but slight contractions of muscles of the lower extremity could be demonstrated. The second sympathetic block was followed by the same degree of improvement evident after the first block. The improvement was transient in the upper extremity but persisted in the lower extremity. On the fifth day following onset of the paralysis, the symptoms indicative of cardiocirculatory insufficiency had subsided sufficiently so that the oxygen tent could be removed. In spite of a satisfactory Horner's syndrome with the sympathetic block on the fourth day following onset of the paralysis, there was no voluntary movement of any of the muscles of the upper extremity except for slight contraction of the adductors of the arm. Slight but unequivocal improvement was evident in the motor activity of all groups of muscles of the left lower extremity. This type of reaction to the sympathetic blocks continued for

the next 5 days. At that point, the patient was able to walk with assistance, and slight voluntary movements of both the flexor and adductor muscles of the arm and of the flexor muscles of the forearm were demonstrated. She was discharged at this stage of convalescence on a program of physical therapy.

Comment: This case has been presented particularly because of the importance that has been attached to the status of cardiac function in the development of cerebrovascular accidents. Twenty-four hours after rather acute cardiac decompensation had developed, suddenly, without pain, the left side of the patient's body became paralyzed but her state of consciousness was not altered. An angiogram was not made to determine if this attack of focal cerebral ischemia was the result of obstruction of the right middle cerebral or the internal carotid artery by a thrombus or embolus or secondary to severe vasospasm. The important point is that in any one of these eventualities, the acute cerebral circulatory disturbance was focal in spite of the possible relationship to the systemic circulatory disorder, that is, the cardiac failure. It also seems extremely significant that, in this 77-year-old woman with cardiac decompensation, cervical sympathetic block should be of any value. However, as indicated previously, the first 2 blocks were followed by slight but definite improvement in motor function in both of the involved extremities. Nevertheless, the definitive reaction to the ischemia consisted of considerable irreparable damage to the involved area of the cerebrum. A recent review[24] of the progress of 107 patients who had sustained cerebrovascular accidents clearly showed that cardiac decompensation at the onset of the stroke foreboded a particularly unfavorable course.

CASE 4.—H. A. K., a 42-year-old geologist, was admitted to Ochsner Foundation Hospital, March 16, 1953, with the complaints of weakness of the right extremities and difficulty in speaking. He was first told that he had arterial hypertension approximately 10 years earlier. He believed that the blood pressure usually was "about 150." He had noted no difficulty that seemed related to the hypertension until 5 months prior to admission when he awakened to find that the left upper extremity was extremely weak and the left lower extremity was slightly weak. The weakness gradually disappeared over a period of about 2 weeks and he returned to work one month after the onset of the weakness. He was told to continue to take one-half grain of phenobarbital 3 times daily "because of the high blood pressure." Approximately 3 months later, while sitting quietly, he suddenly and painlessly became hemiplegic on the right and aphasic. He was placed on a bed and the symptoms began to clear almost at once. He believed they completely subsided in 30 to 40 minutes. Shortly thereafter, he arose but after walking only a few steps, the entire syndrome recurred. Again, there was no headache or other pain. He stated that his "mind was as clear as could be but I just couldn't talk." The blood pressure was reported to have been 170 mm. Hg systolic and 108 mm. Hg diastolic shortly after the last episode. The weakness of the lower extremity rapidly diminished but the patient was concerned about the persistence of rather pronounced weakness of the involved upper extremity and of a moderate degree of difficulty in speaking. He was extremely apprehensive concerning the possibility that another similar attack was imminent.

Neurologic examination on admission to Ochsner Foundation Hospital revealed slight limping on the right as a result of residual weakness of the right lower extremity. The patient had moderate to severe weakness of the muscles of the right upper extremity and slight paresis of the lower muscles of the right side of the face. The musculotendon reflexes were moderately more active on the right than on the left; the latter were normal. The reactions of the superficial reflexes were normal. There was a moderate degree of expressive aphasia. The vessels of the optic fundi were slightly sclerotic but there were no exudates or hemorrhages evident. Proprioception and exteroception were intact. The blood pressure was 162 mm. Hg systolic and 102 mm. Hg diastolic. Clinical and laboratory investigation of the hypertension revealed normal cardiac and renal function and disclosed no cause for the hypertension. The cerebrospinal fluid pressure was 210 mm. of cerebrospinal fluid and its protein content was 40 mg. per cent. It was the consensus that

the patient had had 3 bouts of focal cerebral ischemia secondary to cerebral vasospasm, the first attack having involved the right cerebral hemisphere and the second and third attacks, the left cerebral hemisphere.

On March 19, 1953, superior cervical ganglionectomy was performed on the left with use of a local anesthetic. Within 48 hours moderate to pronounced improvement in the strength of the right extremities and in the ability to speak occurred and persisted. The patient was discharged on the sixth postoperative day. He was readmitted on March 28, 1953, and 3 days later superior cervical ganglionectomy was performed on the right. He again was discharged on the sixth day following operation. On May 12, 1953, he was re-examined and found to have no neurologic deficits except those referable to the sympathectomies, that is, a Horner's syndrome bilaterally. However, he believed that normal strength had still not been regained in his right arm. He returned to work as a geologist in Saudi Arabia 3 months later.

When he returned for re-examination on Oct. 20, 1955, he reported that he had had no further difficulty. He believed that he had recovered completely except for possibly being slightly less able to pronounce certain words as readily as prior to the second bout of cerebral ischemia. The patient's wife stated that this was not evident to her and it was not apparent from the neurologic examination. The Horner's syndrome still was present bilaterally. The blood pressure was 154 mm. Hg systolic and 96 mm. Hg diastolic.

Comment: Within 3 months this patient had had 3 episodes of acute focal cerebral ischemia. The right cerebral hemisphere was involved once and the left twice. We believe that his course following sympathectomy substantiates the conclusion that the neurologic deficits were primarily the result of attacks of cerebral vasospasm. The sympathectomy on the left caused definite improvement in the neurologic deficits, and we believe that the sympathectomies have given him considerable insurance against further occurrence of similar attacks.

DISCUSSION

The problem of acute focal cerebral ischemia may be discussed most satisfactorily from the standpoints of prevention of the attacks and protection of the ischemic brain from ischemic or hemorrhagic infarction after such an attack. Specifically, it is important to realize that although cervical sympathetic block may be a helpful preventive measure and useful initially in the treatment of the focal cerebral ischemia, if the pathologic reaction has proceeded to the stage of impending or actual hemorrhagic infarction, the resulting vasodilatation may worsen the cerebral pathologic alterations. Administration of anticoagulants may be even more hazardous. It, therefore, is obviously important not only to recognize the type of cerebrovascular accident that one is attempting to prevent or treat but also to consider the phase of the pathologic reaction probably present if persistent neurologic deficits are evident.

The lack of agreement regarding the most effective means of preventing acute attacks of focal cerebral ischemia undoubtedly would largely disappear if it were possible to establish the exact cause of the attacks. Such episodes may be partially a problem of hemodynamics, after a fairly adequate adjustment has been made to the primary lesion, such as occlusion of the internal carotid artery, as demonstrated by Case 2 in which the evidence of ischemia in the distribution of the middle cerebral artery on the side of the occluded internal carotid artery developed when the patient assumed a standing position. However, the most common final precipitating factor may be focal

cerebral vasospasm. It is difficult to believe that most attacks of focal cerebral ischemia are due to emboli from atheromatous lesions or from thrombotic reactions about such lesions when a patent vascular tree is frequently demonstrated angiographically or by direct inspection at autopsy.

If an obstructive lesion can be demonstrated at a surgically accessible site, such as the extracranial portion of the internal carotid artery, operative removal of the lesion would be the ideal treatment. Theoretically, the operative procedure might consist simply of removal of a thrombus or atheromatous lesion with reconstruction of the vessel or excision of a segment of the artery with end-to-end anastomosis of the divided artery, insertion of an arterial graft, or anastomosis of the external carotid artery to the internal carotid artery distal to the site of occlusion.

The observations of Paterson[36] relative to the possible relationship of attacks of acute focal cerebral ischemia to angiospasm caused by intimal hemorrhages are of much interest. If this is a common mechanism in the development of acute focal cerebral ischemia, use of anticoagulant drugs as a means of preventing further attacks would seem inappropriate. On the other hand, Paterson[36] found evidence that thrombi may develop opposite intimal hemorrhages of cerebral as well as of coronary vessels; therefore, it might be postulated that anticoagulant therapy could be advantageously directed toward this phase of the reaction.

Denny-Brown[11] pointed out that cerebrovascular accidents commonly occur in patients whose circulation to the heart, kidneys, and often the extremities is already reduced to a critical level and suggested that certain measures, such as use of vasodilator drugs directed toward improving the cerebral circulation, may instead cause additional complications by producing undue ischemia in other areas. Of course, it is entirely possible that administration of vasodilator drugs may cause sufficient generalized superficial vasodilatation with concomitant reduction in systemic blood pressure so that the blood supply to the brain is reduced further and that of vital visceral areas is jeopardized. The localized action of interruption of the sympathetic impulses to the vessels of the involved hemisphere is one of the advantages of that method in the treatment of an attack of focal cerebral ischemia. Cervical sympathetic block would rarely, if ever, cause sufficient alteration of blood flow to deprive another area of a critical amount of blood.

It seems probable, as suggested by Denny-Brown,[10] that eventually there often is pronounced local vasodilatation in an area of the brain made ischemic by occlusion of an artery by a thrombus or embolus. However, it also seems probable that when the pathologic process has reached this phase, sufficient change has occurred in the precapillary, capillary, and postcapillary areas so that the process will continue to hemorrhagic infarction with restoration of circulation. The importance of interrupting the phase of vasospasm early rather than waiting for the phase of "reactive dilatation" appears rather obvious. Relative to the observations[8, 15, 22, 26] that there are dilatation of the pial vessels and increased intracranial blood flow with anoxemia and elevation

of the carbon dioxide tension of the blood, it is important to remember that the primary goal is to improve the physiologic state of an already compromised area of the brain. To attempt to do so with a noxious stimulus that results in generalized dilatation of the intracranial vessels may actually not significantly alter the state of nutrition of the area of the brain primarily ischemic. It probably is significant that in spite of the fact that the vasodilating action of carbon dioxide has been known for many years, the clinical reports of improvement following its administration to patients with focal cerebral ischemia are particularly scarce. This is in striking contrast to the numerous reports now available regarding the successful use of sympathetic blocks in such cases.

A list of the etiologic factors which, in conjunction with systemic changes usually present that have made the patient vulnerable, may constitute the final precipitating agent for an attack of acute focal cerebral ischemia might include: (1) focal vasospasm, as in Raynaud's disease of the extremities or in migraine attacks, (2) thrombosis of a cerebral artery, (3) cerebral embolism (that is, for example, obstruction of the internal carotid artery or a cerebral artery by an embolus arising from a diseased cardiac valve), (4) inadequate systemic and intracranial hemodynamics as demonstrated by bouts of ischemia in the distribution of the middle cerebral artery on the side of an occluded internal carotid artery, (5) focal vasospasm secondary to intimal hemorrhages in the arterial tree, and (6) intermittent obstruction of the cerebral arterial tree by multiple emboli arising from atheromatous lesions of, for example, the arteries of the base of the brain or from a thrombotic reaction about such a lesion. It is evident that the majority of such patients have degenerative changes throughout the arterial tree that contribute to development of the focal ischemia of the brain and lessen the chances of prevention of attacks by treatment directed toward causing some degree of vasodilatation in the involved cerebral hemisphere.

When such considerations are weighed, it seems clear that relative rest and general supportive measures have an important place in prevention of attacks of focal cerebral ischemia. Furthermore, because of sclerotic changes usually present in the arterial tree and because many patients, after having several attacks of acute focal cerebral ischemia without manifesting clinical evidence of cerebral infarction, may have no more, relatively few such patients should be considered candidates for sympathectomy under local anesthesia. On the other hand, several bouts of such attacks may occur during a period of several years, and it is probable that a series of sympathetic blocks during such a series of attacks is a helpful prophylactic measure and lessens the possibility that cerebral infarction will occur. Also, administration of such vasodilator drugs as nicotinic acid and nitroglycerine probably lessens to some degree the factor of vasospasm as a primary or secondary factor in the development of the localized cerebral ischemia without altering the systemic blood pressure sufficiently to constitute a hazard in this respect. If it is concluded that attacks of focal cerebral ischemia represent emboli from a thrombotic reaction, anticoagulant therapy[14] would seem appropriate provided

it can be given in effective quantities without undue risk. The possible relation of transient attacks of focal cerebral ischemia to interference with blood flow through the common or internal carotid arteries in the neck by a surgically amenable lesion can be determined satisfactorily only by angiography.

Treatment directed toward prevention of cerebral infarction following an attack of acute focal cerebral ischemia is not necessarily that which may be appropriate in the prevention of an attack of cerebral ischemia. The primary goal is to restore adequate circulation to the area quickly enough to prevent irreparable damage to the brain and to the tissues constituting the walls of the precapillary, capillary, and postcapillary portions of the vascular tree. This may be accomplished simply by placing the patient in a supine or Trendelenburg position and instituting measures to increase the systemic blood pressure if it has fallen below the patient's usual level. If the symptoms do not disappear immediately, cervical sympathetic block should be performed at once, for it is at this phase of the reaction that the greatest amount of cerebral tissue may be saved and the danger of precipitating or worsening hemorrhagic infarction of the brain is minimal. If the primary lesion is focal vasospasm, more improvement would be expected than if a major vessel were occluded by a thrombus or embolus. The only source of blood available in the latter eventualities, of course, is through collateral channels. Successful exploitation of these potential sources of blood may, however, be obviated or made difficult by such factors as atherosclerotic changes of the arterial system and anatomic deficits of the circle of Willis. If no improvement is evident or if the clinical manifestations only partially subside, in spite of the development of an adequate Horner's syndrome, the block should be repeated as soon as the Horner's syndrome clears. The technique of cervical sympathetic block is easily perfected and, when done correctly, is innocuous and relatively painless.[25] If the evidence of focal cerebral ischemia has persisted long enough so that, theoretically, hemorrhagic infarction of the brain may be in progress but its presence is not indicated by red blood cells in the cerebrospinal fluid, a series of sympathetic blocks probably still offers the most favorable prognosis. Additional measures that may be indicated include a sedative, if the patient is unduly apprehensive or agitated; a vasodilator drug, such as nitroglycerine or nicotinic acid; and general supportive measures, such as oxygen therapy, a cardiac stimulant, and blood transfusion if indicated by examination of the peripheral blood. After institution of these emergency measures consideration can be given to the desirability of performing superior cervical ganglionectomy or simple section of the cervical sympathetic trunk with the use of a local anesthetic. The condition of such patients can usually be evaluated much more satisfactorily if angiography is performed. The potential advantages and disadvantages of angiography must be carefully weighed in each case. Certainly, the possible usefulness of vascular surgery on the cervical portion of the carotid system cannot be determined unless angiograms are made.

Undue damage to the ischemic brain would obviously be postponed if its metabolic activity were reduced significantly but it seems doubtful that this

would be accomplished by merely confining the patient to bed as has been suggested.[11] Hypothermia undoubtedly accomplishes this goal but the dangers accompanying such treatment would usually contraindicate it unless hemorrhagic infarction of the brain appeared imminent or had occurred.

SUMMARY

Many patients in whom acute focal cerebral ischemia develops present a history suggesting that the final, critical preciptating factor is undue spasm of the arterial vessels supplying the involved area. This conclusion is supported by studies at autopsy of patients who had presented such syndromes but demonstrated no organic obstruction of the vascular tree. Furthermore, there is rather complete agreement that with mechanical obstruction at some point of the arterial tree supplying the brain, there is initially some degree of vasospasm distal to the obstruction that interferes with prompt development of the maximum collateral circulation. It, therefore, should follow that cervical sympathetic block should be considered to constitute a potentially effective therapeutic measure. Most authors who have given cervical sympathetic block an adequate clinical trial in such patients have presented generally favorable reports. Careful distinction should be made between treatment directed toward prevention of an attack of focal cerebral ischemia and that directed toward salvaging ischemic cerebral tissue to which persisting neurologic deficits are referable. Four cases of acute focal cerebral ischemia have been presented in which response to interruption of the sympathetic impulses to the involved cerebral hemisphere was favorable to some degree.

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Recent Advances in Surgery

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CLASSIFICATION AND TECHNIQUE OF FASCIAL SUTURES IN THE REPAIR OF INGUINAL HERNIA

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THE successful surgical management of inguinal hernia presupposes that proper attention be given three basic considerations: (1) the type of hernia present, (2) the selection of a suitable technique consonant with the existing anatomic deficiency, and (3) the judicious choosing of appropriate suture material. Since autogenous sutures may also function as anatomic replacement in the formation of a living connective tissue bridge between the anchored parietal wall and the adjacent inguinal or subjacent Cooper's ligament, we have elected to consider suture material as a separate phase in this discussion.

TYPE OF HERNIA

Much information can be gained as to the type of hernia that is present by careful examination of the inguinal canal, but a precise diagnosis ordinarily can be made only at operation. Only after the sac is completely dissected and totally visualized, so that its anatomic relationship is fully evident, can an accurate diagnosis of the hernia be made. The size of the sac, the number of locules present, the extent of the associated mural weakness, and the availability of reliable adjacent structures for the selective reparative procedure all respectively play proportionate roles in the over-all evaluation of the hernia and its surgical management. Too often, there is a tendency for the surgeon to assume an unrealistic complacency toward hernia and the results of hernia repair. In general, the choice and ultimate success of the reparative procedure is dependent as much upon how well the surgeon weighs the contributing links in the anatomic chain which jointly or consecutively contrived to produce the herniation, as upon the suture material employed in the repair. Unless one is especially interested in the academic aspect of herniology it is probably better to consider the classification of hernia as (1) exclusively saccular (indirect), (2) medial sacculation with mural weakness (direct), and (3) combined indirect-direct sacculation with attenuation of the posterior fascial wall forming a bilocular sac. It is the direct component of the hernial sac that is susceptible to eversion through the femoral ring (inguinofemoral).

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SELECTION OF REPAIR

With the acceptance of this simplified classification of inguinal hernia the primary or basic techniques of hernia repair can be reduced to simple exsection of the sac, used only when a narrow tubular process of peritoneum has persisted permitting a small protrusion through the abdominal ring, as in the congenital hernia of infancy and childhood. Appositional sutures in this technique (herniotomy) are few. Actually, no repair of the posterior wall is necessary. The second progressive operational step consists in the removal of the sac and the creation of a first line of defense by suturing the relaxed parietal strata to the recurved portion of the inguinal ligament with simultaneous transposition of the spermatic cord. Sutures in this technique play an important role. The success of this operation rests upon two maneuvers, the exsection of the sac and the synthesis of the apposed wall to the inguinal ligament. Collectively, the operative variants of this classic procedure are referred to as an inguinal ligament hernia repair (herniorrhaphy). The third progressive reparative step consists essentially in anchoring the combined parietal wall to Cooper's ligament. This basic technique has also undergone many modifications. In order to follow the modern trend in terminology and for the purpose of simplification, the technical maneuvers of this operation are generally referred to compositely as Cooper's ligament hernia repair (hernioplasty). As performed in our clinic, the triple maneuver Cooper's ligament repair[3] anatomically is a combined inguinopectineal hernia repair. Its purpose is to take advantage of certain mechanical principles and anatomic features which may derive from both the inguinal ligament and Cooper's ligament procedures, respectively.

CHOICE OF SUTURE MATERIAL

The solidity of union of the apposed structures is essential to the success of any reparative operation for hernia. For instance, in inguinal ligament hernia repair the parietal wall must heal solidly to this ligament to form a protective and resistant shelf; in a Cooper's ligament hernia repair, one or more strata of the wall must unite firmly to Cooper's ligament. The purpose of the suture is to provide a medium for attaining structural union.

The function of sutures may be twofold: (a) that of mechanically holding the apposed structures in the desired relationship until healing is complete (nonviable sutures such as silk, wire, or catgut serve this purpose) and (b) that of tissue replacement, additive to fixation of the adjoined structures. The latter is illustrative of a dual function of sutures and introduces a new concept in the plastic repair of hernia. Fascial sutures augment the effectiveness of the operative procedure, particularly where there are varying degrees of structural deficiency and where tension may be superimposed on the suture line.

The proposed evolutionary classification of fascial sutures is simple, practicable, and chronologic and will be followed in the elucidation of the various technical variants (Table I).

Seelig and Tuholske,[39] Gallie and LeMesurier,[12] Maxeiner,[27] Morse and Barb[28] became convinced that silk sutures as an appositional holding force are

TABLE I. CLASSIFICATION OF FASCIAL SUTURES

I. Autogenous			
A. Pedicled			
1. Single:	McArthur	1901-1904	
	Seelig	1927	
	Carscadden	1934	(interligamentous closure)
	Payne	1935	
	Bisgard	1939	
	Joyce	1940	
2. Double:	McEachern	1924	
	Robins	1938	
	Sachs	1939	
	Gaston	1947	
3. Triple:	Rice and Strickler—1948		
4. Multiple Interrupted:	Hodgkins—1928		
B. Free			
1. Single:	Gallie and LeMesurier—1921 (usually single)		
2. Multiple:	Masson	1935—Fascia	
	Foster	1936—Fascia	
	Burton	1941—Fascia	
	Brown (peritoneum)	1930	
	Levering (peritoneum)	1935	
	Davidson (sac as sutures)	1936	
II. Heterologous, Heterografts, or Zoografts			
	Koontz	1926	
	Rosenblatt and Meyers	1928	
	Egan	1935	
	Chandy	1946	
III. Homologous, Homografts, or Isografts			
	Usher, Morris and Sell	1954	

often insufficient to attain a lasting and unyielding union and therefore the likelihood of recurrence is greater, particularly in those instances presenting a deficiency of the posterior fascial wall. In their experience gained from clinical studies, these authorities found that autogenous fascial sutures possess certain cardinal features which make them superior to silk. Simultaneously, experimental and clinical observations of Koontz,[20] Rosenblatt and Meyers,[36] and Egan and Chandy[6] have shown comparable results by using preserved fascia. Koontz[20] also contends that the areolar membrane of the parietal strata acts as a barrier to solid union by the formation of a thin scar which stretches when stress is applied and insists on its meticulous removal. We, too, would like to re-emphasize the importance of carefully dissecting away any areolar tissue from the appositional surfaces.

It has been shown that autofascial sutures possess superior qualities when compared with sutures of silk, cotton, and nylon. These inherent qualities are: (1) they are elastic and have great tensile strength; (2) they remain viable (autogenous); (3) they are nonirritating to the tissues in which they are imbedded; (4) they may be used for anatomic replacement; (5) owing to their size, ribbon shape, and property of elasticity, they serve to cushion intra-abdominal force and reduce the incidence of tension necrosis; (6) the results of most statistical studies show a higher curability rate of hernia; and (7) fascia will unite with fascia under tension (Law).[21]

Nageotte[29] has observed four distinct phenomena in transplanted fascial grafts. The implant attaches itself to the host by the ingrowth of fibroblasts. A vascular network invests the graft. The cells of the grafts are rehabilitated in their former histologic setting (integral survival). The dead connective tissue cells of a preserved graft are replaced by living cells (reviviscence).

The predominant opinion of the clinical aspects of living versus preserved fascia may be stated as follows. Autogenous sutures produce no tissue reaction, whereas some reaction has been consistently observed, often minimal, in the tissues surrounding the imbedded preserved graft. Autogenous fascia has greater elasticity and tensile strength than preserved fascia (Gratz).[15] Live tissue is more readily transformed into tendinous tissue (Haas[16]). There is integral survival of autogenous fascia (Gallie and LeMesurier[12]), but without metaplasia (Rehn[33]).

Indications for Fascial Sutures.—Where need exists for a viable resistant union of connective tissue or in the presence of structural deficiency, the use of fascia is imperative as in the following conditions: (1) inordinately large indirect hernias with parietal weakness or agenesia usually with loss of obliquity of the canal; (2) direct hernias with attenuation of the fascia transversalis; (3) most recurrent hernias; (4) sacless and some parasaccular sliding hernias; (5) large femoral and other interligamentous hernias; (6) arborization of the sac with multiple exits; and (7) generalized or regional mesenchymal weakness (asthenic).

After having ascertained that one or more criteria for the employment of a fascial suture are present, another equally important decision must follow. It concerns the number of fascial sutures which will be required, or whether because of the extent of the anatomic gap, an inlay graft is essential for its replacement. Since there are varying degrees of weaknesses and deficiencies of the ligamentous and mural structures which often coexist, it is essential that the surgeon be familiar with the methodology of fascial sutures for attaining solidity of the posterior wall of the inguinal canal.

During the first half of the twentieth century there have accumulated numerous reports detailing the various modifications of fascial suture technique. In reviewing these successive evolutionary technical steps there has developed two generally recognized fundamental types of repair, inguinal ligament hernia repair and Cooper's ligament hernia repair. The former, which is bidimensional, alters the level of the wall by placing it at the inferior edge of the inguinal ligament and is the more superficial repair. The operation was popularized in Europe and in the United States almost simultaneously. This technique is applicable chiefly to those patients having fascicular weakness associated with mild hypoplasia of the conjoined aponeurosis. Assuming the presence of a strong inguinal ligament, a fascial suture may be employed to augment any deficiency that may exist in the conjoined aponeurosis in the medial angle of the canal.

Cooper's ligament technique is a deeper repair, actually intrapelvic. It is tridimensional in scope; and, consequently, this operation forms a stronger posterior wall, but decidedly alters the former relationship of the parietal wall to the

trihedral space. In the first maneuver of this repair the trilaminar wall is anchored to the vertical portion of Cooper's ligament. In this operation, the reinforcement of the lateral transitional angle is a crucial step and for which we prefer a fascial suture. In the second step, the inguinal ligament is turned down and attached to Cooper's ligament. The former acts as a pedicled graft by bridging this vulnerable interligamentous space. The converging aponeurotic and ligamentous surfaces are apposed superadjacent to Cooper's ligament as the third maneuver.

Pedicled Single Fascial Suture.—(Fig. 1, McArthur). Unanimous recognition is given McArthur[24a] (1901) for his ingenuity in conceiving and executing the original idea of employing a pedicled fascial suture in hernia repair. Incidentally, McArthur also used a pedicled double fascial suture in 1904.[24b] But, in general, the single pedicled suture technique is the classical operation of McArthur. It is recalled that Cooper made his noteworthy contribution to our knowledge of anatomy of the inguinal region one century antedating McArthur's important innovation.

Technique: The original procedure of McArthur remains basic for all pedicled sutures. Therefore, the description of the initial or parent technique of cutting and placement of the fascial strip will suffice for its many subsequent variants whether they are single or multiple. A strip about 0.7 cm. wide is cut from the superior edge of the external oblique aponeurosis but is left attached inferiorly to the pubic spine. It is threaded on a needle and used as a continuous suture to appose the conjoined tendon and fascia transversalis to the recurved portion of the inguinal ligament. If there is deficiency of the conjoined aponeurosis, the autogenous suture is woven back and forth forming a mat to serve as replacement of the fascial gap. The medial flap of the external aponeurosis may be used to superimpose the underlying fascial network creating a joint fascioaponeurotic wall. Generically, the living suture apposes fascia and some fascicular fibers to fascia. Where some of the fibers of the internal oblique are picked up in the lateral bites of this suture, the perimysium forms the intermediary link in attaining fascial synthesis. Therefore, the first line of defense created by the fascial suture possesses the inherent qualities of great tensile strength, elasticity, viability, and a wide surface all-fascial contact without compromising the function of the donor stratum. It was assumed at the time of introduction of the fascial suture that its nourishment was provided through the pedicle, which was responsible for maintaining its viability. However, this concept of sustaining the nutrition of the fascial transplant subsequently proved to be untenable.

The McArthur fascial suture technique, although a milestone in hernia repair, never gained great popularity. Seelig and Chouke,[37] 1923, and later Seelig,[38] 1927, led the way in proposing new methods to strengthen the closure of the defect in the floor of the canal by means of autogenous fascia. Carscadden,[5] 1934, reinforced the interligamentous space with a running pedicled suture of the external oblique aponeurosis between the shelving portion of the inguinal ligament and the horizontal portion of Cooper's ligament. Patey,[30] 1949,

strengthened the reconstituted internal ring using a strip of the posterior lamella of the rectus sheath. Payne,[31] 1935, proposed another variant, that of employing Cooper's ligament as basic anchorage of the parietal wall in augmenting the floor of the canal by utilizing a pedicled suture of the external aponeurosis. The last modification is presently the preferred technique by many surgeons, except a free fascial suture is employed.

In the appraisal of the McArthur fascial suture technique perhaps the most valid of the alleged criticisms is the restricted length of the suture. In order to compensate for this shortcoming the surgeon is prone to take longer stitches which predispose to chinks and inadvertently create potentially vulnerable areas. In our experience with 156 repairs using the pedicled single suture technique,[4] the recurrence rate in this follow-up series was 4.5 per cent compared to 457 silk repairs with 7.5 per cent recurrences. Joyce,[18] 1940, reported on 337 fascial suture hernia repairs with 6 per cent recurrences for less experienced and 3.92 per cent for the more mature surgeons.

Pedicled Double Fascial Sutures.—(Fig. 1, McEachern, Robins, Gaston.) McEachern,[25] 1924, Robins,[35] 1938, and Gaston,[14] 1947, proposed variations in the dual fascial suture technique. For instance, McEachern,[25] followed the McArthur principle, using the median aponeurotic flap as the donor of the first suture but the other fascial suture was raised from the aponeurosis of the internal oblique muscle slightly superior to the abdominal ring. The latter was applied to the upper portion of the canal for reinforcement of the area subjacent and medial to the abdominal ring. Robins[35] preferred to use both the superior and inferior edges of the external oblique aponeurosis as donor sites of the sutures with each pedicled near the pubic tubercle through the superior and inferior crura respectively. The first fascial suture contributed by the superior edge of the aponeurosis is passed through the rectus sheath at the pubic angle and picking up the opposite ligamentous fibers overlying the pubic bone, with the cord making its exit through the first loop. The latter should not be drawn taut enough to constrict the cord (Fig. 1). The suture continues lateralward apposing the internal oblique muscle to the inguinal ligament. The second fascial suture is started lateral and inferior to the cord which closes the edges of the aponeurosis. The spermatic cord is not transposed. Gaston[14] employs a double external aponeurotic suture; one is pedicled inferiorly, the other laterally. In Gaston's modification, the internal oblique is first apposed to the inguinal ligament with interrupted silk sutures after which it is reinforced with a fascial suture. The cord is usually placed subaponeurotically. The double fascial suture technique greatly augments the strength of the muroligamentous union but coincidentally may create either a gap between external oblique flaps or an inordinately taut apposition of these flaps. As a result of the tension, intra-aponeurotic healing of the reapposed flaps may be jeopardized. Judging from a review of the recent literature, dual pedicled sutures are rarely employed. An attenuated external oblique stratum constitutes a contraindication to the use of this technique. It is, therefore, evident that with these imposed limitations the practicability of this method is appreciably restricted.

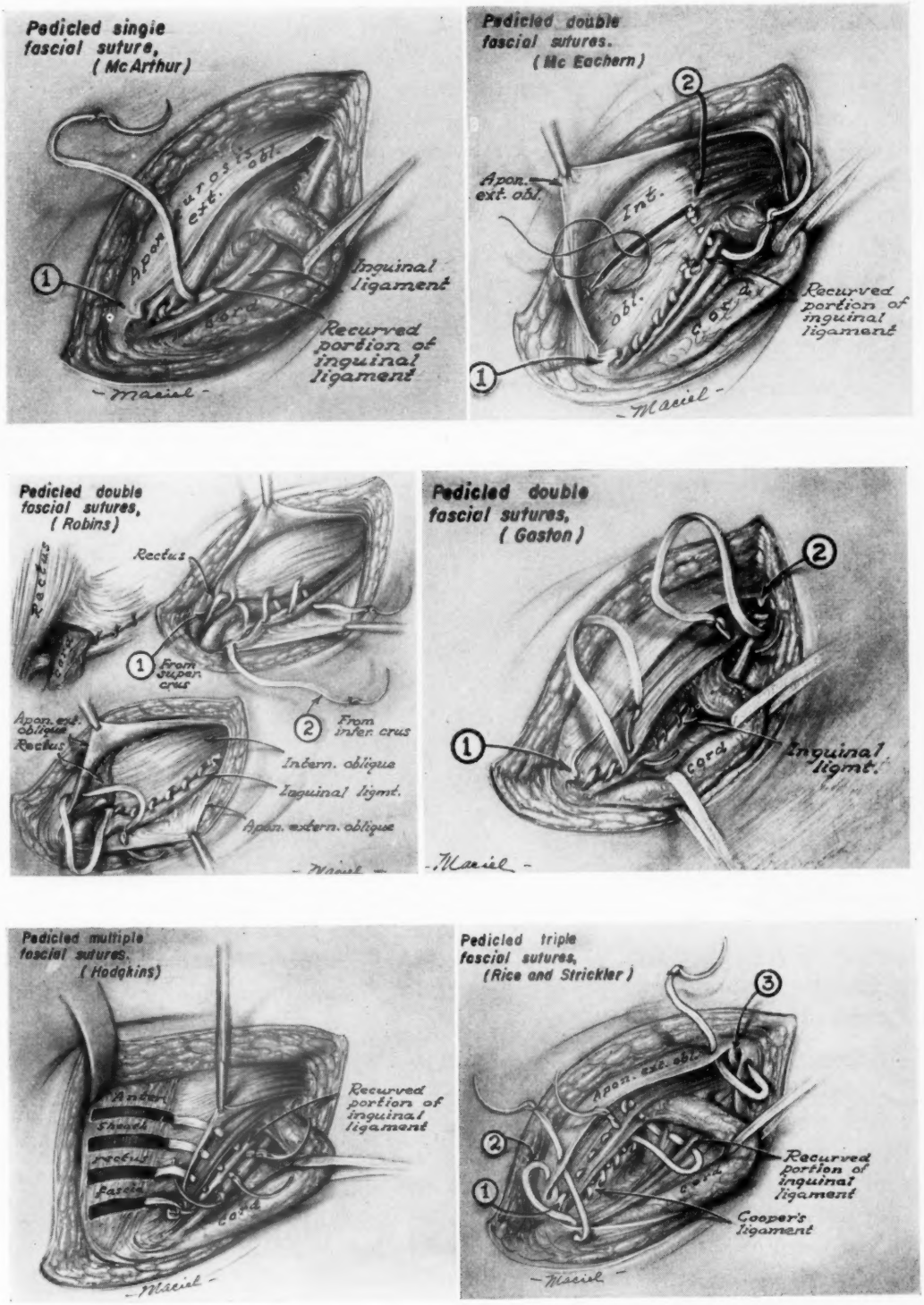

Pedicled single fascial suture, (McArthur)
Inguinal ligament
Recurved portion of inguinal ligament
Pedicled double fascial sutures. (Mc Eachern)
Recurved portion of inguinal ligament
Pedicled double fascial sutures, (Robins)
From super. crus
From infer. crus
Intern. oblique
Inguinal ligmt.
Apon. extern. oblique
Pedicled double fascial sutures, (Gaston)
Inguinal ligmt.
Pedicled multiple fascial sutures. (Hodgkins)
Recurved portion of inguinal ligament
Pedicled triple fascial sutures, (Rice and Strickler)
Recurved portion of inguinal ligament
Cooper's ligament

Fig. 1.—Pedicled fascial sutures, single, double, triple, and multiple.

Pedicled Triple Fascial Sutures.—(Fig. 1, Rice and Strickler.) The idea of utilizing three pedicled sutures in hernia repair was simply a natural evolution of the single and subsequently the double fascial suture concept. What is amazing is the protracted interim of nearly a half century after the report of McArthur's monofascial suture technique before triple pedicled sutures were introduced. After this maneuver was subjected to more mature and meticulous surgical analysis it was found wanting in that it demonstrated the fact that there is a limit to the number of fascial sutures that can be feasibly employed without sacrificing the function of the important external oblique as a stratum. Rice and Strickler,[34] 1948, reported their experience with the triple pedicled suture technique, but I have not been aware of any subsequent articles dealing with this modification of compounding the McArthur principle.

Pedicled Multiple Interrupted Fascial Sutures.—(Fig. 1, Hodgkins.) Instead of employing the external oblique aponeurosis, as an intrinsic stratum of the inguinoparietal wall as the donor structure, Hodgkins[17] conceived the idea of going adjacent and superior to the inguinal area. He raised a series of interrupted pedicled sutures from the rectus sheath transversely to the long axis of the rectus muscle. These fascial strips are left attached to the lateral edge of the sheath. Each is threaded on a needle and carried through the shelving portion of the inguinal ligament. For that matter, Cooper's ligament is equally applicable for their anchorage. Because of their shorter length, many sutures are required to adequately reinforce the floor. The advantage of this procedure is that the donor structure is not compromised; the former site of the strip regenerates rapidly and the intrinsic parietal strata of the inguina remain intact. There are disadvantages to this technique also. It is time consuming. Unless the sutures are closely placed, weakness and intersuture protrusion may occur. It has been pointed out by Chouke[7] through anatomic research on the sheath of rectus that it is variable in density and that the level of the linea semicircularis is not only inconstant in different individuals but there is often unilateral inconstancy in the same individual. Hodgkins' technique, while unique, is rarely used in the repair of inguinal hernia. However, we believe it is useful and we have employed it successfully for bridging midline abdominal defects. In the latter, both recti are used as donors for the fascial strip.

Free Autogenous Fascial Sutures.—(Fig. 2, Gallie, Burton, and Davidson.) Through increasing experience, certain distracting features of the pedicled suture were becoming manifest, such as: (1) attenuation of the external oblique aponeurosis, which affects its density and texture; (2) multiple sutures contributed by intrinsic anatomic structures of the inguinal area which constitute a predisposing factor to recurrence by substituting a possible weakness of one or more of the donor structures for the primary mural defect; and (3) inconstancy of the aponeurotic component, often limiting the strength of the suture obtainable. These objections to pedicled sutures gave impetus to the possibility of substituting free fascial sutures. The latter, if found to be feasible, could be procured from several sources remote to the operative site.

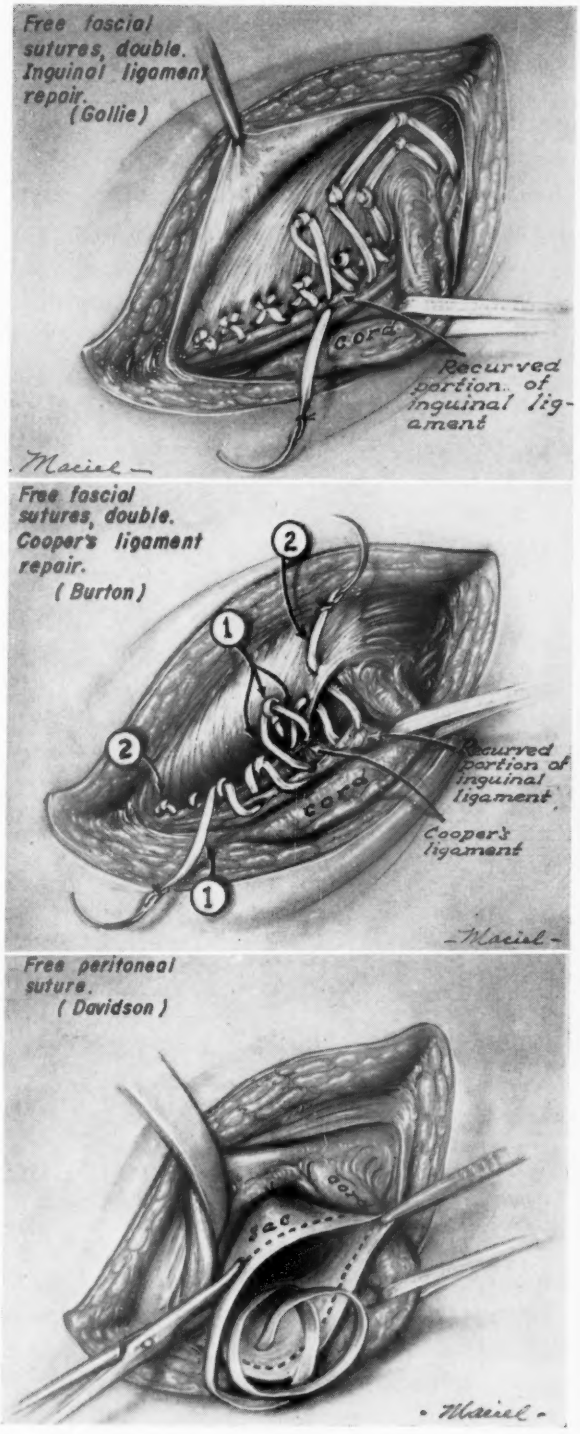


Fig. 2.—Free autogenous fascial sutures, double, single, and peritoneal.

In hernia fascioplasty it is well to review the long-recognized basic principles which have evolved in tissue transplants. (1) The transplant must be capable of living or regeneration. (2) Its implantation must be in proper environment. (3) It must function. (4) It must be generically related. The fulfillment of these criteria by Gallie and LeMesurier[12] through their experimental and clinical achievements using free autogenous fascial sutures gained for them international renown. Moreover, they established permanently the status of the free fascial suture in reconstructive hernial operations. Fortified with this additional knowledge of the fate of the living fascial suture and the reported success with free fascial sutures, many clinics accepted the newer concept which in turn resulted in increasingly wider clinical application of the free fascial suture technique. Incidentally, it had been shown by Kirschner's[19] experiments, which antedated the research of Gallie and LeMesurier, that fascia is different from other body tissues in that it derives its nourishment from the lymph surrounding it and not so much from its blood supply. Consequently, the inevitable conclusion drawn from Kirschner's studies is that the pedicled fascial suture possesses no advantages over a free fascial suture.

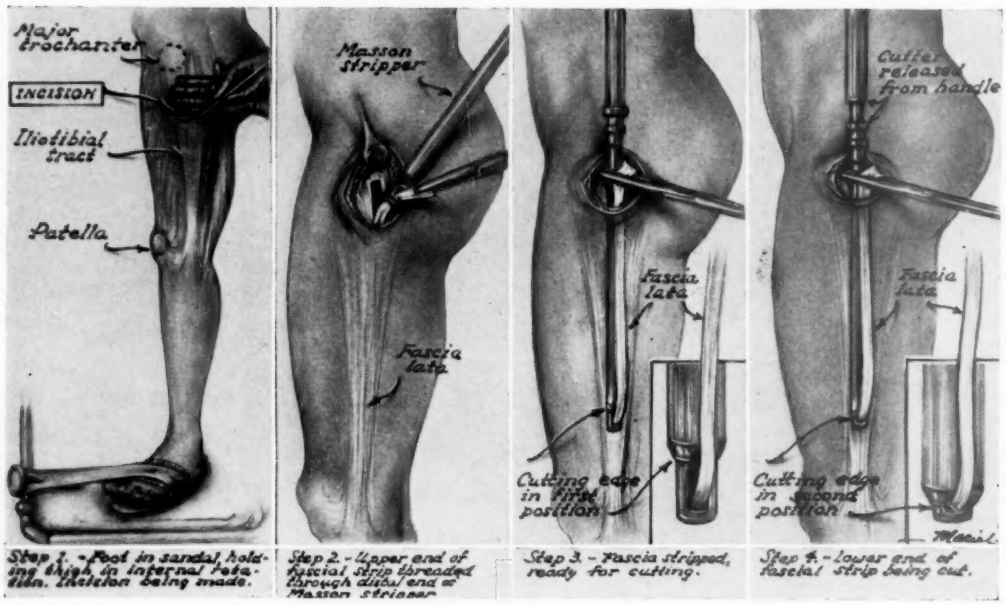

Major trochanter
INCISION
Iliotibial tract
Patella
Step 1. - Foot in sandal, holding thigh in internal rotation. Incision being made.
Masson stripper
Fascia lata
Step 2. - Upper end of fascial strip threaded through distal end of Masson stripper
Fascia lata
Cutting edge in first position
Step 3. - Fascia stripped, ready for cutting.
Cutter released from handle
Fascia lata
Cutting edge in second position
Step 4. - Lower end of fascial strip being cut.

Fig. 3.—Masson technique for extracting fascia lata strip.

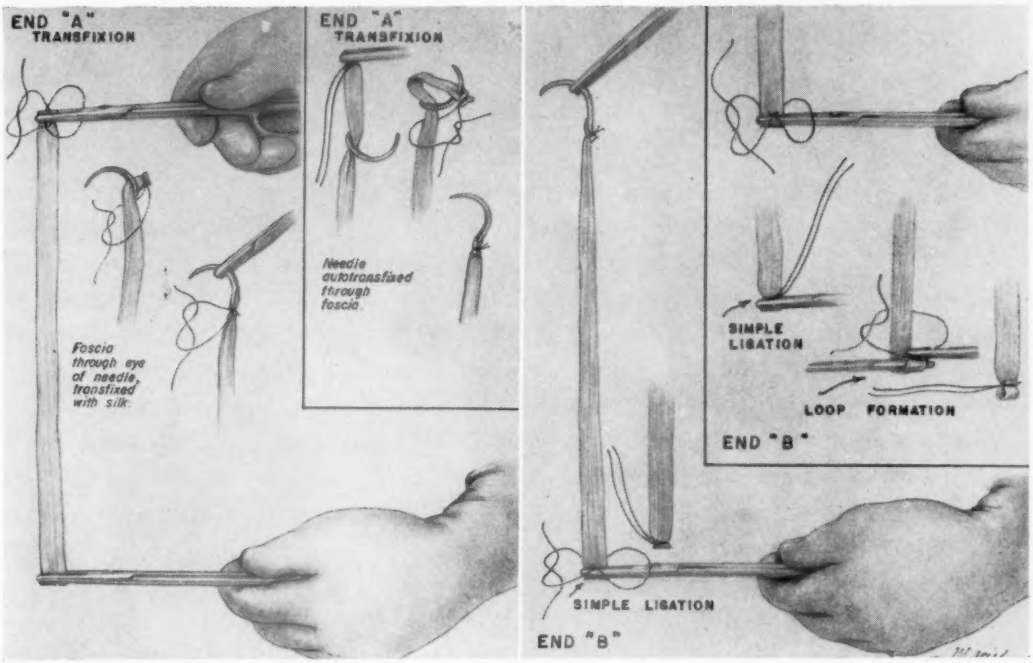
Free autogenous fascial sutures may be used singly or multiply. The fascia lata became the initial source of fascial strips and has continued to constitute the principal donor structure. It possesses the advantages of constancy and easy accessibility and affords longer and denser strips of fascia than is possible from the external oblique aponeurosis or sheath of rectus. If there should be need for multiple sutures they may be obtained from the same donor structure without additional exposure. Through the ingenuity of Masson,[26] an instrument was devised which greatly facilitates the extraction of the fascial strip. The

technique of removing a strip of fascia lata preparatory to employing it as a suture is very simple (Fig. 3). It has been previously described and illustrated by Masson[26] and by Burton.[3] Normally, with the patient relaxed and supine, the thigh assumes the position of external rotation. It is difficult to parallel the direction of the fascia lata with the thigh in this position. In forcing the fasciotome down the normally externally rotated thigh, there is shearing of the strip before reaching the inferior level of the fascia. Too often this results in a short and pointed strip. To correct the external rotation of the thigh, a sandal has been devised (Fig. 3) which has a sole plate and is covered on its dorsum with an elastic band. The latter feature makes it adjustable to any foot. The sandal is applied to the foot after the thigh is rotated internally and holds it in this position by a 2-inch-wide canvas strip extending from the sole of the sandal around the dorsal aspect of the foot, which then is attached to the opposite side of the operating table. By this simple gadget, the thigh is maintained in internal rotation which facilitates the passage of the instrument parallel to the fascia lata without its veering out of line of the long axis of the thigh. A 2.5 cm. transverse skin incision is made four fingerbreadths below the base of the greater trochanter. The latter is constant and is an easily recognized landmark. In our extensive experience in the extraction of 1,500 fascial strips, the upper limit of the fascia lata which is at its junction with the tensor fascia femoris has remained constant. A small gauze sponge 2 cm. in diameter placed on a hemostat serves as a very good dissector for wiping away the adherent areolar tissue, exposing the underlying shining fascia. A fascial strip, about 1 cm. in width, is raised 2.5 cm. and then threaded through the lower end of the Masson stripper (Fig. 3). The stripper is held parallel to the thigh and firmly pushed toward the patient's knee, at the same time holding the fascial strip taut with a hemostat, until a strip perhaps 20 cm. in length is freed. The outer tube of the instrument is unscrewed a couple of turns from the handle, releasing it for cutting the inferior end of the strip. If another strip is desired, and ordinarily two strips are employed, the procedure is repeated taking a second strip adjacent to the first one.

For a time after the introduction of the free fascial suture technique, there was some apprehension as to the possibility of the development of a herniation through the fascia lata donor site. But, suprisingly enough, on careful follow-up examinations a muscle hernia almost never occurred. The reason for this remained obscure until Foshee[10] demonstrated three layers of the fascia lata, an inner transverse layer, an outer transverse layer, and a strong middle longitudinal layer. The inner and outer layers regenerate, are in continuity with, and are direct extensions of adjacent corresponding layers of normal fascia lata which invariably proceed to regrow to span the defect. Owing to the rapid metaplasia of the transverse layers it is unnecessary to suture the edges of the fascia lata. In contrast to the transverse layers, the longitudinal layer of the fascia lata does not possess the property of regeneration; consequently, it is not possible to obtain a suitable fascial strip that can be used as a suture by restripping the former donor site.

Preparation and anchorage of free fascial sutures: (Fig. 4.) In the adaptation and practical application of the free fascial suture to hernia repair, it is important to condition our thinking in terms of the experience gained from its predecessor, the pedicle suture. The basic or primary differences in the two sutures is that the latter retains its initial or normal anatomic fixation at one end, whereas in the former some means must be devised to securely anchor the other free end. The transfixion of the proximal end of the fascial suture (End "A") to the Gallie needle is the same whether the suture is pedicled or free. The ligated end of the fascia is threaded through the eye of the needle and tied securely so as to overlap itself perhaps 0.6 cm. (Fig. 4, End "A" transfixion through eye of needle). The troughlike depression at the base of the needle adjacent to the eye, in effect, reduces its thickness due to reduplication of the suture. Gallie employed this type of transfixion and it is the one most commonly used. It is important that the fascial suture be of proper size so as to follow the needle tract without tearing the ligamentous structures through which it passes. The other method of transfixion of the fascial suture to the needle is to draw the ligated end through the eye perhaps 3 cm. until the point of the needle will pierce the fascial suture immediately proximal to the ligated end, which produces a slip knot when drawn taut (Fig. 4, End "A" needle autotransfixed through fascia). The latter transfixion maneuver has been described and illustrated by Masson, Robins, and Gaston. It is the method of choice for fascial strips 0.7 cm. or wider. In narrower strips, the first or Gallie method is preferable, as the needle does not pierce the fascia and, therefore, would be less likely to tear out of the eye during the process of the insertion of the fascial suture and the step which immediately follows, that of pulling the suture taut. There are two recognized maneuvers for fixing the free distal end of the suture after its ligation. The needle is passed through the fascial strip juxtaproximal to the ligated end which in effect acts as a slip knot (Fig. 4, End "A" through End "B," single transfixion). This maneuver is simple to perform and is very satisfactory when there is not inordinate pull on it because of tautness of the apposed walls. Where most of the pulling force on the approximated wall is transmitted through a series of continuous stitches, this method has been found to be very satisfactory. The other alternative method of transfixing the distal end of a free fascial suture is to form a loop, perhaps 0.5 cm. in diameter, by turning the ligated end back on itself and transfixing it circumferentially with silk (Fig. 4, End "A" through loop "B"). The loop transfixion is much stronger and is preferred when there is reasonable expectation that considerable force may be applied to the suture line. The loop method is especially indicated where there is greater focal tension, as, for instance, at the lateral junctional angle of a Cooper's ligament repair. Having been convinced of the additive strength of the loop maneuver, we have routinely adopted it for the first or transitional angle suture in Cooper's ligament repair. The fact that in the formation of the loop there is slight shortening of the suture is of no consequence. It is all-important that the vulnerable part of Cooper's ligament hernia repair is securely fixed to Cooper's ligament by the most reliable and

available medium, a living fascial suture. Such a suture has been observed to resist greater pressure from the apposed walls as, for instance, when the patient suddenly strains when under too light anesthesia. There is reason to believe that some unrecognized recurrences take place during the immediate



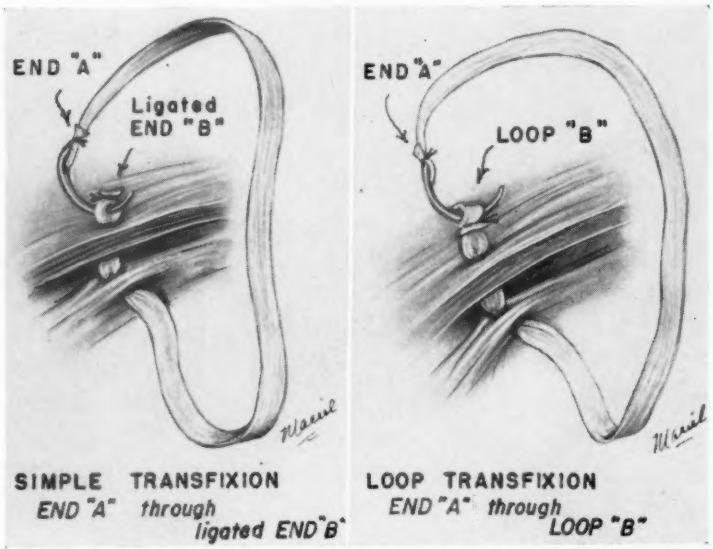


Fig. 4.—Preparation and anchorage of free fascial sutures.

postanesthetic period when the patient is retching, which often superimposes inordinate strain on the suture line of the newly reconstructed wall. We have seen silk sutures break under such stress. Observation of fascial sutures subjected to similar strain retained their integrity.

It might be apropos to call attention to the fact that in pulling up the slack after placement of the continuous fascial suture that each successive loop be pulled taut, starting at the beginning of the suture and progressing toward its distal end. The placing of a curved hemostat, such as a gall-bladder forceps, under each successive loop and lifting on it is a simple and effective means of attaining uniform tautness to the apposed structures. The surgeon is cautioned to avoid attempting to pull up the slack in the suture for its entire length by exerting inordinate force on the needle. Such thoughtlessness often results in the suture pulling out of the eye of the needle.

The anchoring of the detached needle end of the fascial suture while important has caused us little concern. We have been unaware of any slippage of a suture. The end of the suture is transfixed with silk on completion of the last stitch. We have not found it necessary to use a half-stitch or to autolock the suture until reaching its end. Gallie has illustrated a third method which consists of splitting the end of the fascia and tying the strands around the suture proximal to the last bite in the fascial wall. Experience has shown that it is seldom necessary to reinforce each stitch with transfixion sutures of silk. The placing of a second row of fascial sutures paralleling and remote to the first one or darning of the vulnerable area of the posterior wall is illustrative of the different methods of fascial reinforcement depending upon the extent of the tissue deficiency. The fascial strip should not be allowed to dry before it is used. If the suture is placed in a solution of normal saline for long it becomes edematous. To maintain normal hydration of the fascial suture it should be placed in a slightly moist saline sponge until ready for use. It is the usual practice not to proceed with the removal of the fascial strip until the operative field is made completely ready to start the repair.

Technique of Cooper's Ligament Fascial Suture Hernia Repair.—(Fig. 5.) When the criteria for the employment of fascial sutures occur, Cooper's ligament repair is considered the operation of choice. This technique comprises almost 20 per cent of the more than 5,000 hernial operations performed at the Veterans Administration Hospital, Dayton, Ohio, in the last 20 years. The operation consists of three maneuvers. In the first maneuver the combined parietal strata forming the medial boundary of the canal are attached to the vertical portion of Cooper's ligament. This suture line extends from the external iliac vein to the pubic spine. One fascia lata strip is required for the completion of this step. The inguinal ligament is next turned down and its edge is anchored to the anterior surface of Cooper's ligament. The second fascial suture which is used for this maneuver passes through the loops of the first one and consequently does not penetrate Cooper's ligament. With the completion of the second maneuver there is created a V-shaped depression bounded by the converging walls, which have been attached to Cooper's ligament. The adjacent surfaces of the superior and inferior mural components of the repair are next apposed by retrogressive continuation of the second

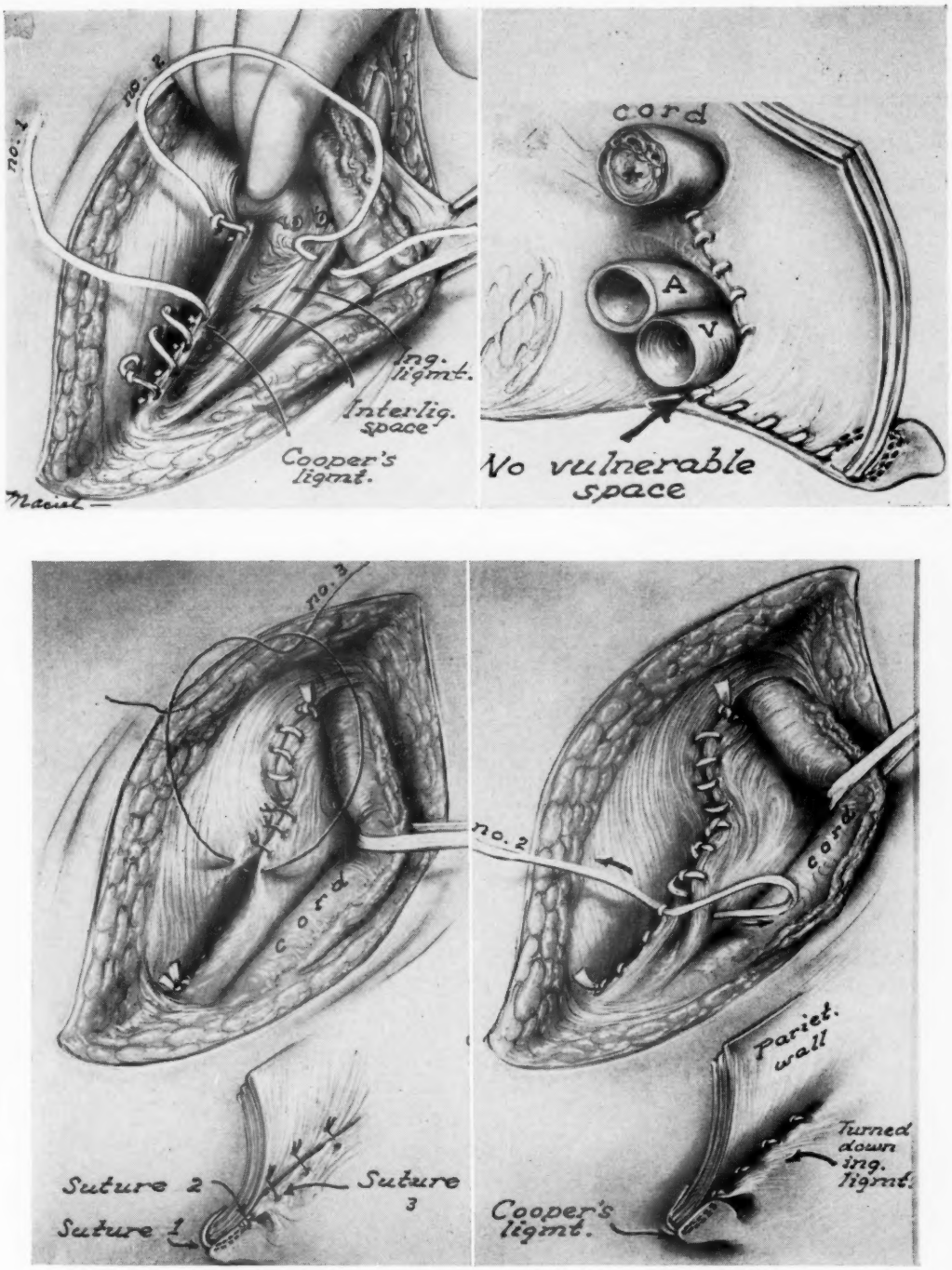


Fig. 5.—Technique of Cooper's ligament fascial suture hernia repair. *A*, First maneuver. *B*, Cross section of first maneuver. *C*, Second maneuver; inset, cross section. *D*, Third maneuver; inset, cross section.

fascial suture or by using interrupted silk sutures. Actually the third maneuver integrates the first and second maneuvers by producing a synthesis of the inverted pectinealized walls.

The conjunctive action of the three maneuvers incorporated in this operation results in greater security because of the dual Cooper's ligament anchorage of the parietoligamentous walls, which is further augmented by the synthesis of the two mural components. From statistical studies of 726 Cooper's ligament fascial suture repairs followed for 1-, 5-, and 10-year periods, respectively, the recurrence rate was 2.25 per cent. In a series of 334 hernial repairs using identical technique but substituting silk sutures, the recurrence rate was 5.7 per cent or double that of the fascial sutures. Garner[13] reported recurrence of 2.5 per cent in a series of 555 operations using fascial sutures (Gallie technique). In the same report 298 hernias were repaired with catgut with 10.1 per cent recurrences, and 1,459 silk repairs were performed with 7.3 per cent recurrence. These follow-up data point convincingly toward the superiority of fascial sutures in hernia repair.

Other sources of free fascial or connective tissue sutures include the plantaris tendon and the peritoneum from large hernial sacs. Pilcher[32] employed plantaris tendon grafts in hernia repair and contends that they possess advantages over fascia lata in being easier to handle surgically, more easily calculable as to length, and stretching laterally to form a stratum. The latter feature renders it ideal for hernia repair. Its disadvantages are its agenesia in 7 per cent of the cases, and the tendon may be of inadequate size and strength in an additional 9 per cent of the cases. Daseler and Anson[8] found that in approximately one third of those persons in whom the muscle is wanting it is bilaterally absent. Because of these objectional features we have employed this tendinous suture in perhaps only a half dozen patients in a total of 6,000 hernia repairs in our clinic.

A living peritoneal suture obtained from strips of the hernial sac has been used in the repair of hernias by Brown,[1a] Davidson,[9] and Levering[23] (Fig. 2). There are, however, certain prerequisites of the sac which must occur to make it suitable as suture material, such as its thickness and size in order to give adequate suture length and strength. Davidson[9] who determined the comparative strength of peritoneal and fascial sutures was convinced that the former had sufficient tensile strength to be utilized in the repair of certain inguinal hernias. Similarly, it has been shown that the peritoneal suture will unite with fascia and with the connective tissue elements of muscle. In recent years, large hernial sacs are rarely encountered so that the possibility of obtaining peritoneal sutures from this source is rapidly becoming obsolete.

DISCUSSION

The primary objective of hernia repair has not changed since the introduction of the modern concept of its surgical treatment by Bassini and Halsted. However, the proper therapeutic management of difficult hernias has continued to plague the surgeon and subsequently led to the introduction of fascial

sutures. A composite panorama of some of the evolutionary maneuvers directed toward the perfection of the fascial suture technique has been presented. Apropos of this technique three factors should be emphasized. (1) The anatomic deficit of the posterior boundary of the canal must be of such severity as to require a fascial suture to attain a lasting repair. In our experience about every sixth hernia will fall in this category. (2) Proper anchorage of competent adjacent or transposed structures is essential. (3) Anatomic and physiologic balance must be maintained in the revised structural adjustment.

Interest in the use of fascial sutures has fluctuated widely. In some clinics there has been considerable enthusiasm for the fascial suture technique, which is usually traceable to the investigative spirit of a few surgeons striving for progressive improvement in their reparative results. However, in general, there has been paucity in accepting the fascial suture concept. The principles of the fascial suture technique were not accepted earlier for several reasons. The surgeon failed to familiarize himself with the various sources and methods of obtaining fascial strips. Not infrequently the need for fascia had not been anticipated and consequently the donor site had not been prepared. Such a preparational departure at this stage of the operation often means breaking of aseptic technique and confusion of the operating team. To obviate the occurrence of the latter in every patient in which a fascial suture might be indicated, the donor field should be prepared at the same time as the inguinal area. Operating time is increased. While this factor may have been valid initially with the development of the fasciotome, fascial strips may be obtained with no significant delay in completion of the repair. Simplification in the preparation of the fascial suture and in its transfixion during its placement have contributed to shortening of the operating time. The false concept that wound infections are significantly higher with fascial sutures was no doubt a deterrent to the acceptance of this technique. With the observance of recognized aseptic precautions, maintaining careful hemostasis, and gentleness in handling of the tissues, infections should be no higher than in other reparative procedures. Indecision arising from the lack of a clear-cut understanding of the criteria for the use of fascia has perhaps been an adverse factor at times. With an increasing number of articles appearing in the literature, there has been concomitantly wider use of fascial sutures, which is gradually overcoming the former receptive lag of this technique. In some of the earlier reports, the incidence of recurrence was higher with fascial suture repairs.[1b] However, there are presently adequate cumulative data on the fascial suture regime[5, 10, 12-14, 18, 22] to negate the impression gained from former statistical studies. Integral survival of the autogenous suture was not universally accepted at first. But, any doubt was resolved by the excellent research of Gallie and LeMesurier. We have had the opportunity to re-examine autogenous fascial sutures 2, 3, 6, and 22 years, respectively, following their initial placement. In each, the suture remained macroscopically unchanged. Histologic study of three of the sutures revealed viable fascia. Clinically there was convincing evidence of the viability

of the fourth fascial suture, but, unfortunately, the removal of a segment of the suture for histologic confirmation was not feasible. An additional clinical feature of a hernial fascioplasty worthy of mention is the greater resistance of the repaired wall which is readily demonstrable on the follow-up examination. This uniform solidity is especially recognizable when the parietal wall has been pectinealized.

Fascial suture variants, whether pedicled or free, have progressed through successive experimental and clinical stages. In this collective review, the more significant ones have been illustrated. Obviously, some of the methods have become obsolete but they shared in contributing invaluably to our knowledge of fascial suture technique. Conjunctively, fascial sutures have expanded the horizon of hernia repair. Hernia, primarily, is a local entity, but, its repair is often regional in scope. Occasionally hernia may be a systemic manifestation, as in mesenchymal weakness. If the possibility of hernia repair were thought of as regional in scope at the time of the initial operation, particularly the difficult ones, there is reason to believe the recurrence rate would be lower than presently exists.

CONCLUSION

1. A collective review with a practical clinical classification of fascial sutures has been presented.

2. Standard techniques employing fascial sutures have been evaluated and illustrated.

3. Clinical and experimental research has led to a better understanding of the destiny of the living and preserved fascia. The latter has become obsolete.

4. Extraction of the fascial strip with the fasciotome and its preparation for employment as a suture has been described and illustrated.

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Editorial

CONDUCTED BY I. S. RAVDIN, M.D.

The Stethoscope—Historical Instrument?

Recently I was informed of a clinician displaying a stethoscope in a small glass case with the legend beneath, "Historical Instrument." Although this was done with some degree of humor, it serves to focus the increasing abandonment of the stethoscope in the diagnosis and management of surgical patients.

True, the ever-increasing development of laboratory and technical equipment has largely diminished previous reliance on auscultation. There still remain a host of circumstances in which there is no substitute for a stethoscope. This is best exemplified in the detection of the "silent abdomen." Absent peristalsis may result from a number of pathologic conditions. When diagnosis of an important lesion above the diaphragm has been ruled out, a "silent abdomen" may be prima-facie evidence of peritoneal irritation, suggesting surgical intervention. This is frequently experienced in challenging diagnostic problems where the clinical picture is obscure and the laboratory findings are misleading.

All surgeons have experienced the concern and dilemma encountered in acutely ill and/or unconscious patients with possible ruptured viscus (not confirmed by x-ray in 15 per cent of the cases), subcutaneous abdominal injuries, vascular accidents, and the whole gamut of inflammatory lesions. Listening through a stethoscope placed on the abdomen enables one to determine the nature of peristaltic activity. Skillful interpretation of such auscultatory findings may prevent a needless operative delay in some patients and, on the other hand, spare other patients unnecessary surgery.

Close postoperative attention to the abdomen and chest demands meticulous use of the stethoscope and bears no further comment. Lastly, thorough auscultation may reveal unsuspected lesions of the neck, chest, abdomen, and extremities in routine examinations.

Great technical advances have been made since Laennec described the stethoscope 140 years ago. The stethoscope still remains an important clinical tool transcending all specialties—a symbol of the art of medicine. This is more adequately expressed by the eloquent words of Sir William Osler, "Nor can I claim to speak for pure science, some sort of which remains from early association and from a lifelong attempt to correlate with art, a science which makes medicine."

—*Joseph R. Wilder*
Baltimore, Md.

Appraisal of Progress in Surgical Therapy

CONDUCTED BY WARREN H. COLE, M.D.

COLECTOMY WITH SIMULTANEOUS ILEOSTOMY AS THE SURGICAL TREATMENT OF DIFFUSE ULCERATIVE COLITIS

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SOME controversy over the surgical treatment of ulcerative colitis still exists among present-day surgeons and internists. There is general agreement that the diseased bowel must be removed before the patient can be entirely well, but there is still some wavering opinion as to whether ileostomy should be done as a preliminary step to colectomy. Undoubtedly the fantastic morbidity and mortality of the past is still in the minds of many.

Before proceeding with the discussion, it might be well to find out where American surgeons stand on this issue. Because of the relative rarity of the disease (and in comparison to the surgical treatment of duodenal ulcer), few surgeons have had extensive personal experience with the surgical treatment of nonspecific ulcerative colitis; consequently, the number of men who would express a personal opinion is rather limited. Rosi[21] stated that in recent years because of improved surgical techniques and aids he has employed a one-stage procedure much more frequently than heretofore. He felt that any patient who required an ileostomy because of severe universal disease of the colon should have the colon removed at the time the ileostomy was made. He stated, "In toxic disease it is especially desirable to remove the diseased colon at the time of ileostomy because in this way the patient is relieved of the basic cause of his symptoms." Baker[1] also feels that there is a definite advantage in carrying out colectomy with simultaneous ileostomy rather than ileostomy as a preliminary stage. He stated, "The one-stage operation (ileostomy with colectomy) is most desirable in the toxic stage and has its advantages in treating most of these patients." Ripstein,[19] after a large personal experience, advised immediate colectomy with ileostomy as the treatment of choice in all patients requiring surgery for ulcerative colitis or its complications. He noted that the mortality and morbidity in this type of surgery was greatly lowered since he had taken a more aggressive attitude toward primary colectomy. He stressed primary colectomy with ileostomy as the procedure of choice in the treatment of the acute fulminating phase. Scarborough[22] also continues

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to perform colectomy with simultaneous ileostomy—in fact, he favors proctocolectomy with ileostomy whenever possible to reduce the morbidity factors associated with the stage procedures. Cave[8] is of the same opinion after a very long association with the disease. The opinions and practices of these surgeons reflect a definite change from those of the past. Past practices will be discussed later, but before attempting to analyze the reasons for recent trends we must consider the views held by other well-known American surgeons with a long and continuing experience with ulcerative colitis. Cattell[5] has recently noted a change in the operative procedures for ulcerative colitis that were employed at the Lahey Clinic. He notes that at this time the majority of their cases have primary subtotal colectomy with simultaneous ileostomy as a first stage, followed in 3 months by abdominoperineal resection. He stated that they had performed a few total colectomies with ileostomy in one stage. His present opinion of the surgical approach to the acute fulminating phase of the disease is different from the ones just listed, however. He stated, "Fortunately, we are seeing fewer of the acute fulminating type and I think it is in this group that our opinion would differ from yours, for we would still do ileostomy alone in these cases." Dixon[13] cautioned against a one-stage proctocolectomy in very ill patients. He noted that he preferred to carry out an ileostomy as a first-stage procedure, followed by subtotal colectomy because of the fact that ulcerative colitis is so often a systemic disease. In reference to removal of the colon and rectum with the establishment of an ileostomy in one stage, he stated the following, "It seems to be the thought to carry out this procedure, close the patient tightly 'fore and aft,' and have it all over within a short space of time. If I have a point, it is that in very ill patients you may have it 'all over with' quite suddenly after such a major procedure. My appraisal on this subject may be entirely incorrect. My feeling, however, is that in severe cases I prefer ileostomy first and the colectomy secondarily."

It does not take much analysis to show that controversy exists mainly over the surgical approach to the fulminating toxic phase of the disease. As usual, the roots of such a controversy are buried deeply in the results of the past.

EVOLUTION OF THE SURGICAL TREATMENT OF ULCERATIVE COLITIS

Nonspecific ulcerative colitis is a dangerous disease and people die of it. In 1895, W. Hale White,[27] physician to Guys Hospital in London, in describing his experiences with ulcerative colitis, stated, "The prognosis is grave," and "We have no convincing evidence that the disease is recoverable." He described the autopsy findings accurately. Although the internists of the day had little to offer besides "Bed rest, hot fomentations, and a slop diet," surgery was unthinkable until Weir,[26] in 1902, reported appendicostomy as a means of irrigating the colon with antiseptic solutions. Stubborn cases of amebic colitis were much improved, but the treatment was abandoned because of the failure of the nonspecific ulcerative colitis patient to respond. In time ileostomy (diversion of the fecal stream) became widely used in the hope that

"tranquilization of the colon" would lead to healing of the disease. The results were so bad in most cases that surgeons issued the plea for earlier ileostomy before structural changes had occurred in the colon. The results of ileostomy alone were particularly disastrous in the toxic fulminating group. When confronted with critically ill toxic patients, many surgeons would not even perform ileostomy. But in patients selected for this procedure, the 50 per cent operative mortality was considered "justifiable." Table I lists the mortality from ileostomy alone when carried out in the acute fulminating toxic form of the disease. Ileostomy as the treatment for chronic forms of the disease resulted in an imposing list of complications and in eventual mortality. Surgeons were loath to "maim the young" and to remove the colon in the event that the disease might unaccountably regress. After a 10-year trial of ileostomy, Cattell[6] came to the conclusion that it was not often possible to re-establish continuity. He was able to close only 14 ileostomies of 145 patients, and 6 of these had to have the stoma re-established later. Eight did well for 6 months to 8 years.

TABLE I. MORTALITY FOLLOWING ILEOSTOMY IN ACUTE TOXIC FULMINATING ULCERATIVE COLITIS*

Cave and Thompson[9]	53.3%
Cattell and Sachs[7]	66.0
Rankin[18]	31.7
Kunath[15]	75.0
Bargen, et al.[2]	54.0†
Average	50.5

*Reprinted by permission of Gastroenterology, 19: 58, 1951.

†Thirty-four per cent died within 14 days postoperatively and an additional 20 per cent died within 6 months.

By 1948, there appeared to be more favorable progress in the surgical treatment of ulcerative colitis. Although there was a general appreciation of the permanent curative effect of removing the diseased bowel, it was removed piecemeal in stages, with many hospital admissions, and an accumulated morbidity and mortality that is hard to forget. In 1943, some stirring and radical thoughts in regard to the problem began to appear. Sir Hugh Devine[12] apparently became convinced that ileostomy was of little value with the disease in situ. In describing his treatment of 2 "desperate" cases, he said, "No half measures such as appendicostomy, cecostomy, or even enterostomy are of any permanent avail in these patients. Nothing short of colectomy will save such a patient's life." He did not, however, carry out primary colectomy with ileostomy and aptly summed up the opinion of the day by remarking that colectomy under such circumstances would fail because of the patient's poor general condition.

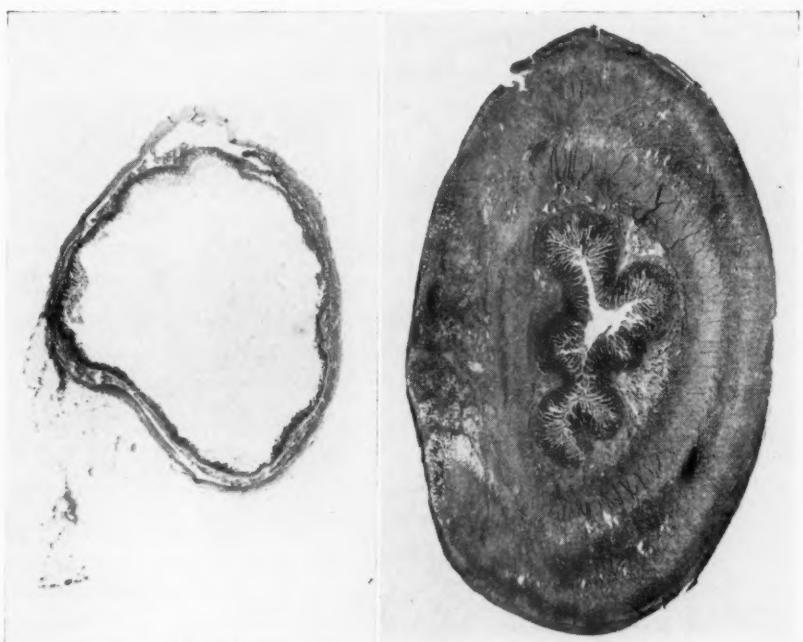
A bold step into the present-day concept of the surgical treatment was taken by Ferguson and Stevens,[14] in 1948. They reported 2 critically ill patients with the toxic fulminating form of the disease upon whom they had carried out subtotal colectomy as an emergency procedure. In each case a previously made ileostomy had failed to alter the course of the disease, whereupon colectomy was immediately carried out with prompt recovery. Each

patient was toxic, had bled excessively, and had perforative disease. They coined the refreshing phrase "Too sick not to be operated upon." In 1949, Miller and his associates[16] reported a small group of patients undergoing primary subtotal colectomy with ileostomy. Shortly after, Ripstein and his coworkers[20] reported the results of subtotal colectomy with simultaneous ileostomy in 72 patients. There were only 3 deaths, all of which could be attributed to failure to remove even more of the disease. They noted troublesome complications which were due to the ileostomy and the ever-present sepsis.

We see, then, that regardless of ileostomy as a preliminary step, the role of the diseased colon-in-situ was appreciated. There was accumulated experience at hand to suggest that most of the colon could be removed (in one stage) without undue risk in chronic forms of the disease and that the same operative procedure was most effective in toxic disease. Within a few months, Crile and Thomas[10] had carried out subtotal colectomy with simultaneous ileostomy in 7 critically ill patients in the toxic phase. Although there was one death of a patient who was moribund at the time of operation, they stressed the fact (and we must continue to stress this) that operation *was not withheld in any case* because of the condition of the patient. It must be noted that until this time, the surgeon called upon to see the critically ill and toxic patient could select those he considered most likely to survive an ileostomy alone. The deaths in the untreated group were not considered to be surgical mortality. This is in contrast to our present concept in which *every critically ill toxic patient* is operated upon whenever medical treatment has nothing to offer. Crile and Thomas[10] also described what has proved to be the most valuable guide to the surgeon for operative intervention—the progressive dilatation of the colon as demonstrated by physical examination and flat films of the abdomen (Fig. 1).

Finally, a better understanding of the pathologic physiology of the conventional ileostomy[23] has led to a change in technique[11] which has lowered mortality and morbidity even further. Until 1951, the technique of ileostomy was not altered in principle from that of John Young Brown.[4] Either the end or a loop of ileum was exteriorized. Diarrhea, weight loss, and fluid and electrolyte problems that followed in most patients were ascribed to the sudden loss of the absorptive function of the right colon. Actually, in the light of subsequent observations, we have found that the serosal surface of the conventional ileostomy became the site of severe inflammatory changes (Fig. 2). Edema, rigidity, and lack of propulsive peristalsis in the ileostomy itself resulted in functional obstruction of the small intestine with the production of the fluid and electrolyte problems already mentioned. The intestinal obstruction produced in this way was particularly intolerable in the wasted and bad-risk patient leading to additional operative procedure with prolonged morbidity and an appreciable mortality[25] and still further complications such as obstructive ulcerative ileitis with perforation and hemorrhage.

Fig. 1.—Colon ileus: the sign of impending perforation in the course of toxic ulcerative colitis.



A. *B.*

Fig. 2.—Cross section of an ileostomy. *A*, Normal intestine, day of operation. *B*, Massive edema with cellulitis (serositis), seventh postoperative day. (Reprinted by permission of J. A. M. A. **158**: 32-34, 1955. Copyright, 1955, by American Medical Association.)

Finally, the bold attack on this disease in its most violent and toxic form may well have been impossible prior to the use of antibiotics and prior to the time when blood and concentrated human serum albumin were not available as they are at this time. There is no question that the modern surgeon can do "what it is possible to do." Because of the work of physiologists and chemists, the technical advances in materials and equipment continue to make the surgical possibilities even greater.

TOXIC UNIVERSAL COLITIS

I would add my emphasis to the opinion of most surgeons; in universal colitis, colectomy with simultaneous ileostomy is indicated in every case where other management has failed. But, primary colectomy is particularly urgent in toxic disease or when any other state of emergency exists, such as hemorrhage or perforation. Since a number of people in the toxic phase recover, prolonged medical treatment in the face of odds is understandable. Therefore, we are going to continue to be asked to carry out colectomy on patients who are in the worst possible condition. There is no way to "build up" such a patient except by promptly removing the disease. At this Clinic more of these patients are coming to surgery than in the past. Twenty-one of the last 53 colectomized patients were operated upon in an emergency status. We attempt to have every colitis patient with fever and tachycardia attended by the surgeon while under intensive medical treatment so that he may exert his judgment where colectomy becomes necessary. He must also have the following information prior to colectomy: (1) the status of the circulating volume so that blood replacement may be made prior to operation; (2) determination of the albumin fraction of the plasma protein; (3) the preoperative treatment status with steroidal drugs; (4) sensitivity history to antibiotics; and (5) preoperative stool cultures for enteric pathogens, particularly *Micrococcus pyogenes aureus.*

The medical (antibiotic) treatment of ulcerative colitis results in a marked depression of the colonic flora. These people are long in hospital residence and have low resistance and altered bacterial antagonisms; therefore, they are particularly susceptible to postoperative staphylococcal enteritis. For example, 9 of our last 57 colectomies for ulcerative colitis were so affected, and one expired with an overwhelming staphylococcal jejunitis. For these reasons preoperative rectal cultures for pathogenic micrococci (staphylococci) may be of great value. The cultures should be made on blood agar and phenyl ethyl alcohol. Any significant growth of coagulase positive staphylococci may help the surgeon anticipate a postoperative enteritis. The signs of onset of staphylococcal enteritis are between the second and seventh day and are fairly constant, with abdominal distention (ileus), pain or cramps, and almost concomitant tachycardia. Diarrhea (rice-water stool) from the ileostomy is a fairly positive sign, while a Levin tube is usually necessary to draw off the large volume of gastric and small intestinal reflux present. Immediate oral and intravenous treatment with an appropriate drug will save the patient, but a

preventive antibiotic is not indicated since resistant mutants may overgrow the susceptible organisms, rendering the antibiotic in use (perhaps the most effective one) ineffective.

When subtotal colectomy with ileostomy is done as a one-stage procedure, the blood loss is minimal. However, it is imperative to add at least 1,000 c.c. of whole blood to the circulating volume of these depleted people in the several hours just preceding surgery. This will stabilize the deficient circulation sufficiently to allow colectomy to be performed without fall in blood pressure. We are ever-sensitized to the dangers of blood given in the operating room when it is not absolutely necessary. Errors in handling are more common when it is given under duress by persons not closely associated with the patients. In addition, blood volume replacement during hypotensive states and trauma of surgery is somewhat hazardous. As a rule we do not remove the rectum in toxic patients because of the excessive hemorrhage that may occur in the presence of acute inflammatory disease.

Another hazard in the toxic patient is the lowered albumin fraction of the plasma proteins. We have found a reversed albumin-globulin ratio to be the rule. Serum albumin levels of under 1.5 Gm. have not been unusual and colectomy has been carried out with as little as 0.7 Gm. The principal danger is that of overhydration and rapid diffusion of fluid into the lungs. Two or three units of concentrated human serum albumin are usually given just as the colon is removed rather than to lose it through the ulcerated bowel prior to surgery. One or two units daily postoperatively may be given to bring the albumin fraction to normal.

STEROIDAL DRUGS

Practically every patient coming to surgery has been treated with steroidal drugs. It is important to know how much and over what period of time. Withdrawal during a period of stress may result in fatal collapse. In combating this problem, the important thing to remember is that overtreatment is better than undertreatment. Although steroidal replacement is an individual problem, a good plan to follow is to give 100 mg. of cortisone (by injection) in the immediate preoperative hours, 100 mg. of Cortef (soluble) intravenously during colectomy, and 100 mg. of cortisone (by injection) in the few hours after surgery, and, thereafter, 50 mg. of cortisone every 6 hours for a day or two. Frequent blood pressure values will determine if this dose is adequate. It should be decreased as rapidly as possible and its withdrawal covered by ACTH. We have not seen faulty healing in any case because of these drugs, and separation of the wound edges or dehiscence has not occurred (steel wire closure).

COLECTOMY FOR CHRONIC COLITIS

Removal of the colon during the chronic intractable phase of the disease is relatively simple; the colon is contracted and the splenic flexure is low and does not present much of a technical problem. If the disease is not too acute, the rectum may be removed with the colon and with minimal blood loss by

simply pulling the anus up through the levators until the perianal skin can be cut across. Dissection is kept directly on the rectal muscularis and the levators are gently pushed off the rectum on all sides. Because of loss of body fat stores, the ischiorectal fat spaces are soft and mobile, allowing the anus and perianal skin to be pulled right up into the pelvis. Thus, the entire proctocolectomy may be done through a relatively short left paramedian incision, while ileostomy is made through a stab wound in a previously selected site on the right side of the abdomen. Reperitonealization of the gutters is not necessary; indeed, it may be harmful. Cattell[5-7] has pointed out long ago that the lymph channels and nodes of the mesentery are infected and when incised, liberate myriads of coloform organisms into the free peritoneal cavity; thus any resection for inflammatory disease is far from aseptic, whether the bowel has been opened or not. For this reason we follow the suggestion of Poth[17] and allow 300 to 400 c.c. of 0.5 per cent neomycin solution to flow freely through the peritoneal cavity during colectomy. After proctocolectomy a small laparotomy tape is placed down through the levators and sphincter remnants to control local bleeding. The pelvis is peritonealized at the close of the procedure. This pack is removed under intravenous Demerol on the third postoperative day and we have not had bleeding in any case. One or 2 ounces of 1 per cent neomycin solution is instilled into the presacral space through the sphincters every other day until the tenth day, at which time any remaining blood clots are aspirated through a short sterile proctoscope. Infection in the space has been rare, but can be immediately brought under control by daily catheter lavage carried out by the patient at home.

EMERGENCY COLECTOMY

Colectomy in the toxic patient is done to terminate the disease and to prevent perforation. Progressive dilatation of the colon with local tenderness has already been discussed as the indication for immediate colectomy. Most of the prejudice against primary colectomy is based on just this type of case and for good reason! Not only is it difficult to remove the colon without rupturing it, but walled-off perforations may have taken place already. Most often the disease is acute rather than chronic, and the colon not only is dilated to the extreme but the splenic flexure is thrust higher than normal. The initial incision should extend up from the pubis to the umbilicus, at which point it is carried diagonally across the left upper abdomen to the lateral-most costal margin. Even with this exposure, the colon may be unattainable because of its extreme friability and dilatation. There is almost no peritoneal cavity to work in because in dilating, the colon has expanded into the leaves of its mesentery, thus seriously reducing its mobility. The situation may be obviated by gently passing under direct vision (and with the abdomen open), two No. 36 rectal tubes (attached in tandem) up from below into the descending colon (Fig. 3). Intermittent suction will rapidly empty the left side of liquid and gas, while gentle manual pressure on the cecum and right colon will result in partial collapse of this side of the organ. Not only is exposure

enhanced, but the colon contracts as it is deflated and its walls thicken, with less danger of rupture and spillage. This procedure has been carried out in 9 patients under these circumstances. Because of the extreme inflammatory reaction around the colon, other organs may be intimately attached and appear inseparable. I make special reference to the duodenum, stomach, spleen, and loops of small intestine. It must be borne in mind that each adherent organ may be protecting a full-thickness perforation and before either is disturbed, the colon should be evacuated thoroughly, as described above, and irrigated with several liters of ½ per cent neomycin solution, if necessary. Mobilization of the colon under these circumstances must be done with the greatest of care to prevent rupture. The best plan is to incise the lateral reflexion of peritoneum as close to the bowel as possible, and to "shell the colon out" of its mesentery. The transverse colon and splenic flexure particularly may be dealt with in this fashion. If fecal spillage occurs, peritonealization of the gutters is absolutely contraindicated. Infection is effectively handled in the peritoneal cavity, but not behind it. Large abscesses may form behind the reconstructed peritoneum of the gutters, have no localizing symptoms whatsoever, and lead to death from septic shock. In addition we believe that the ligated vascular pedicles should be trimmed as short as possible and covered briefly with peritoneum to prevent the small intestine from adhering to them. Finally, manipulation of the infected colon causes portal bacteremia. In addition to peritoneal lavage with neomycin solution, the antibiotic of choice in a given case should be administered intravenously during colectomy.

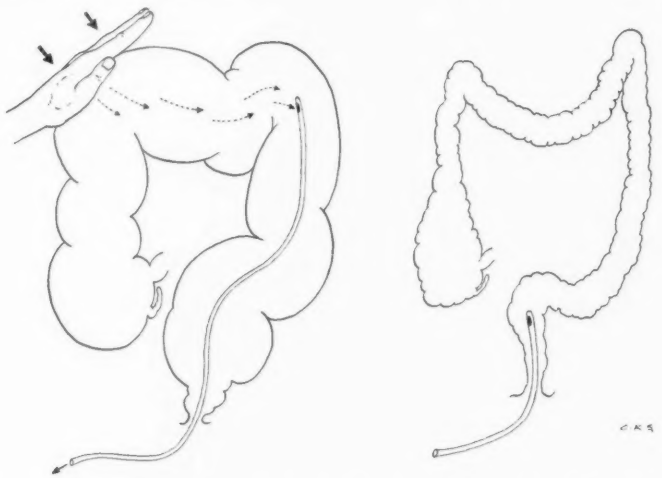


Fig. 3.—Aspiration of colon at time of surgery in toxic ulcerative colitis.

THE ILEOSTOMY

We can no longer see any reason to perform a conventional ileostomy. It places the critically ill and wasted patient at every disadvantage, including the possible loss of life. It results in partial small intestinal obstruction which is intolerable under the circumstances. Obstruction of this type is well tolerated in patients who have not lost any great amount of body weight and

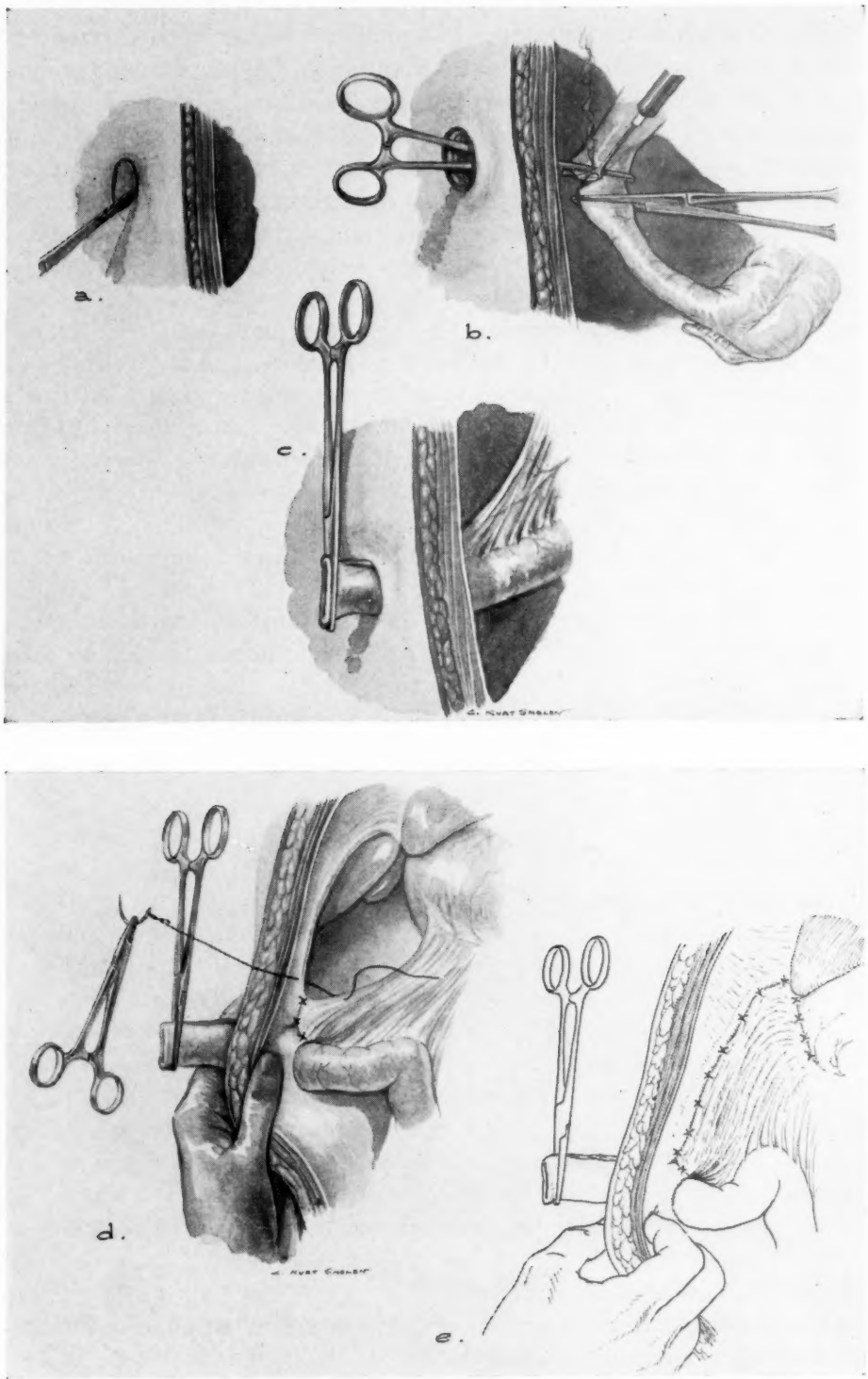
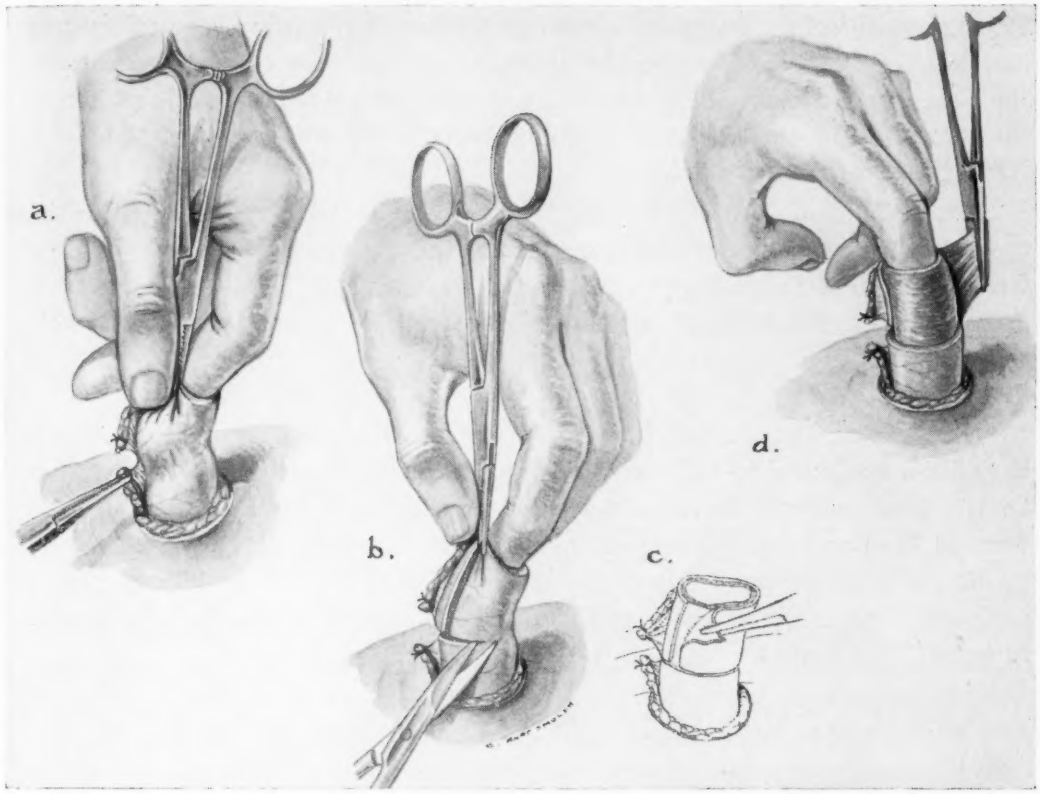


Fig. 4.—Method of ileostomy. (Reprinted by permission of S. Clin. North America 36: 841, 1956.)



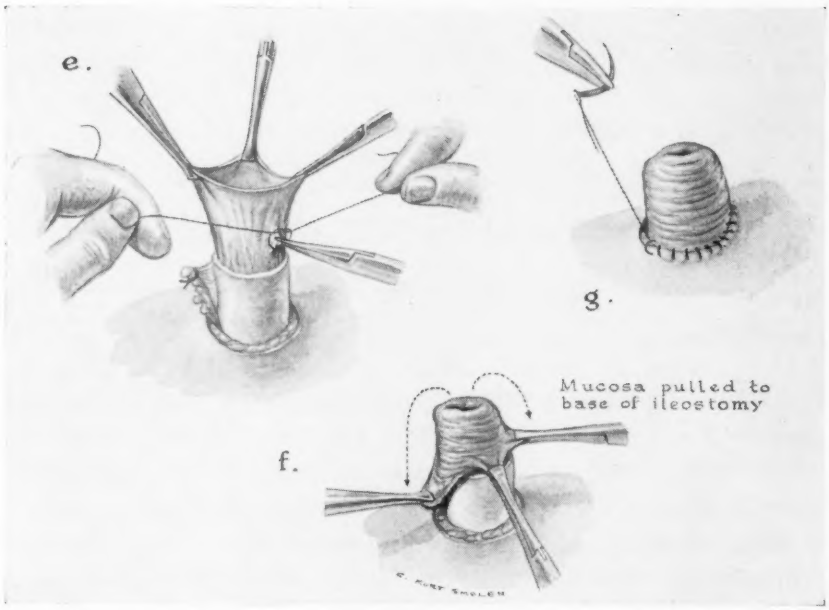


Fig. 5.—Preparation of mucosal graft. (Reprinted by permission of S. Clin. North America 36: 841, 1956.)

while there is time for spontaneous maturation[23] of the ileostomy to take place. The expected weight loss is well tolerated. For this reason every effort must be made for the wasted patient to have a physiologically correct ileostomy at the time of colectomy. Either one of two methods[3, 11] is acceptable as long as the serosal surface of the projecting ileostomy is covered to protect the underlying portion from serositis.

Figs. 4 and 5 demonstrate the technique of ileostomy. After attaching the mesentery to the parietal peritoneum, the abdomen is closed with steel wire. The distal ileostomy (Fig. 5) is demuscularized and the resultant mucososubmucosal remnant is pulled down over the ileostomy and sutured to the skin.

RESULTS

In the past 6 years all patients with universal colitis necessitating surgery have been subjected to colectomy with simultaneous ileostomy. One hundred twenty-three patients have been treated in this manner. Forty-one of these were in the acute toxic fulminant phase of the disease. There were 6 deaths, all in the toxic group, while there were no deaths in the chronic intractable patients. The causes of death are listed in Table II. Sepsis is still the greatest threat to life among these patients.

TABLE II. SUBTOTAL COLECTOMY SIMULTANEOUS ILEOSTOMY

CAUSES OF DEATH—(TOXIC COLITIS)	
Adrenal failure	1
Pulmonary edema	1
Staphylococcal enteritis	1
Unresolved peritonitis	3*

*One was moribund at time of surgery.

TABLE III. SUBTOTAL COLECTOMY SIMULTANEOUS ILEOSTOMY

	NUMBER CASES	HOSPITAL MORTALITY	SUBSEQUENT MORTALITY
Toxic	41	6	
Chronic	82	0	1*
Total	123	6	

*Volvulus at home.

Since 50 patients having subtotal colectomy with simultaneous mucosal-grafted ileostomy have just been followed and reported,[24] it seems opportune to compare the results with an equal number of patients operated upon consecutively during 2 other phases of our surgical evolution: namely, multiple-stage colectomy with conventional ileostomy and colectomy with simultaneous conventional ileostomy. Table IV lists the factors which we believe account for the principal morbidity in this type of surgery. Mortality could hardly be included in this comparison, since the surgeon of Group A selected the toxic patients that he thought would survive an ileostomy alone (the remaining

deaths were medical), while the 2 surgeons of Group B and Group C carried out subtotal colectomy on all toxic fulminant patients not responding to medical therapy.

TABLE IV. COLECTOMY FOR ULCERATIVE COLITIS
COMPARISON OF THREE PHASES OF TECHNIQUE

PROCEDURE	PATIENTS	STAGE OPERATIONS (TOTAL)	ADDITIONAL OPERATIONS ON ILEOSTOMY	ADDITIONAL LAPAROTOMY (COMPLICATIONS)	PERIOD OF OBSERVATION** (YEARS)
A. Multiple stage colectomy with conventional ileostomy	50	184	40	16	7-10
B. Colectomy with simultaneous conventional ileostomy; later rectal resection	50	100	22*	9	3-5
C. Colectomy with simultaneous mucosal-grafted ileostomy; later rectal resection	50	100	6	2	1-3

*Removal of 12 skin grafts.
**Most complications arose within the first year.

The morbidity of multiple-stage procedures is incalculable. Multiple anesthetics, parenteral fluids, and drug administration, nursing care, wound infections, and numerous periods of hospitalization are contributory. The number of reoperations on the ileostomy have been markedly reduced with utilization of stab wound placement and mucosal graft. For instance, most of the reoperations of Group A were for prolapse and conversion of loop-to-end ileostomy for malfunction of one kind or another; skin strictures, bag-fitting purposes, and so forth. By comparison there has been only one prolapse in Group B and none in Group C. We are fairly sure that fixation of the mesentery plus separate placement of the ileostomy through a small opening in the abdominal wall will result in fewer herniations of the ileostomy. Finally, corrective laparotomies for obstruction and sepsis were reduced in Groups B and C because of fewer operations to remove the colon and the use of antibiotics which has prevented peritonitis.

CONCLUSIONS

Primary total or subtotal colectomy with simultaneous ileostomy is the operation of choice in all patients with diffuse ulcerative colitis.

Colectomy with ileostomy is more effective in toxic ulcerative colitis than ileostomy alone.

Morbidity factors are reduced by one- or two-stage operations combined with mucosal-grafted ileostomy.

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Reappraisal

CONDUCTED BY CLARENCE DENNIS, M.D. ASSISTED BY ALFRED HURWITZ, M.D.

ANOTHER LOOK AT THE SUPER-RADICAL OPERATION FOR BREAST CANCER

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THE Editors of the Reappraisal section have asked me to state what my current views are with reference to the super-radical operation for breast cancer. Few operations have had a more difficult struggle. Yet, if the conventional radical operation has a place in the management of breast cancer, then it should be rational to defend the extended operation, of which the major objective is to excise a group of regional lymph nodes in each direction, in which the cancer might extend, beyond those reached in the usual Halsted operation.

The Earlier Operation.—The story of the extended breast operation in this clinic has been one of slow and painful evolution. In July, 1946, when operating upon a patient for cancer of the breast in whom small, hard, shotlike lymph nodes were found to extend upward along the axillary vein, even beyond the point where the vein dipped over the first rib into the thorax, I felt an urge to take down the bony barrier which prevented free entry to the thorax presented by the first rib and clavicle. However, the patient's son, a distinguished physician on the staff of a distant medical school with a long and resplendent record, stood by my side. Such an occasion is not the most appropriate time to undertake aggressive exploratory ventures as many a surgeon has come to know. However, the lingering image on what might have been done was real enough that I promptly suggested to several of my surgical colleagues, more concerned with and expert in operations for breast cancer than I, that we should explore the posibility of enlarging upon the usual radical operation for such cancers. When more than 2 years had gone by and no one in our clinic had yet undertaken to extend the conventional radical operation for breast cancer, I felt that I should try it myself. My first such operation was in November, 1948. In the first 2 patients, the clavicle was divided in its midsection and the medial half was discarded after excision-avulsion at the sternum. The greater length of the first rib was excised and the cartilaginous end of the second rib was cut near the sternum to permit ready excision of the internal mammary artery and vein and the accompanying lymphatic chain of lymph nodes. This exposure afforded the opportunity to perform a supraclavicular cervical and upper

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anterior mediastinal dissection together with excision of the homolateral internal mammary chain of lymph nodes as well as the conventional radical mastectomy. Because the resultant deformity was not fully acceptable to the patients nor to me, I made several attempts in additional patients to preserve the medial segment of the clavicle, employing various types of fixation. All ended in nonunion.

At the meeting of the American Surgical Association in April, 1949, I was able to report upon 5 patients operated upon in this manner with or without preservation of the sternal end of the clavicle. By the time of the next meeting of the American Surgical Association, in April, 1950, I had come to adopt the midline sternal splitting incision, with lateral cutting of the sternum into the first and fourth intercostal spaces, and spoke with a background of experience of only 15 operations. This incision together with excision of the first rib gave marvelous exposure and permitted ready access to the upper mediastinum. All or a portion of the thymus was removed, and the homo- and contralateral innominate veins were cleared. In a few patients the contralateral internal mammary chain also was removed.

However, respiratory difficulties were encountered in some of the patients, engendered in all likelihood by the multiple bony segments created by the nature of the surgical exposure. Several of the patients upon whom this operation was performed were in the eighth decade of life and obviously were not good risks for such extensive procedures. Accustomed to extensive one-stage operations for cancers of the alimentary canal in the interests of a lessened morbidity and mortality, I somewhat reluctantly and uncomfortably settled down to the acceptance of a two-stage operation for the extended operation for breast cancer. In the first stage, the usual Halsted operation was performed, combined frequently with excision of a segment of the first rib. At the second stage performed 4 to 6 weeks later, the supraclavicular, internal mammary, and mediastinal dissections were performed. The two-stage procedure converted the superradical operation into a safe operation which could be done with a mortality not far out of line with that of the Halsted operation. However, it is not necessary to dilate upon the advantages of the one-stage procedure; they are well-known to patients as well as to surgeons.

No operation which is unsatisfying in some respects remains static. Its virtues and defects are being reviewed intermittently with a view toward improvement. The reason the one-stage extended breast cancer operation was not as well tolerated as similar aggressive operations in the alimentary tract seemed always to narrow down to the question of the surgery of access. Over a period of several years, I have had occasion to discuss and correspond with a large number of surgeons who have made important contributions to the extension of the conventional operation for breast cancer. Among others are: Mr. R. S. Handley (London), Dr. J. L. Brenier (Grenoble, France), Professors Erling Dahl-Iverson (Copenhagen), Mario Margottini (Rome), and Pietro Bucalosi (Milan). And among my American colleagues, I should mention particularly Oscar Noel (Nashville), Harris Shumacker (Indianapolis),

Jerome Urban and Irving Ariel (New York), and especially my own local surgical colleagues, Drs. F. John Lewis (now of Northwestern Medical School, Chicago), and S. W. Arhelger and A. P. Thal of this clinic. Dissatisfaction over the two-stage operation and a continuing search for some better variant of techniques previously employed, which would permit readoption of the one-stage procedure—these were the items which continuously recurred in all discussions relating to the nature of an acceptable extended operation for breast cancer. These deliberations and discussions have led to a return to the one-stage operation.

The Present Operation.—A single long oblique to transverse incision is employed which begins beneath the axilla and extends slightly downward, crossing the midline, with elliptical sacrifice of skin over the breast and the neoplasm. The conventional Halsted operation is first carried out. Wide undercutting of the skin flaps permits ready access to the neck and suffices to expose the ensiform cartilage in the epigastrium. The sternocleidomastoid muscle is cut free from the clavicle and the sternal end of the clavicle is disarticulated, permitting the clavicle to be swung outward and either upward or downward as the dissection may require. Employing a Lebsche sternal knife, driven by blows from a wooden or rubber hammer and proceeding usually from below upward, the entire sternum is split in its midline from the manubrium through the xiphoid cartilage. The attachment of the scalenus anticus tendon to the first rib is now cut free, a maneuver which facilitates the plane of the dissection in the direction of the mediastinum as well as the neck. A somewhat more limited exposure is provided by this technique than by that previously employed, in which the sternocostal junction additionally was severed into the first and fourth intercostal spaces. However, the exposure is very adequate for all purposes of the operation.

The advantages of this method of performing the super-radical operation are: (1) it is a one-stage procedure; (2) employment of an oblique to transverse incision eliminates the hazard of necrosis at the lower margin of the medial skin flap; (3) complete splitting of the sternum provides only 2 bony segments which need to be rewired, rather than the multiple segments presented by the midline split of the upper sternum into the fourth intercostal space with an additional break of the costosternal arch into the first intercostal space. This simplification of the procedure insures a stable, bony thorax and precludes respiratory embarrassment.

Detachment of the sternocleidomastoid from the clavicle without excision of muscle permits an adequate cervical dissection (submohyoid); this maneuver, together with fixation of the clavicle in its normal position by a few capsular sutures, virtually does away with any suggestion of deformity. Placement of the lateral end of the skin incision below the axilla serves to preclude contractures which delimit the range of motion of the arm.

In order to perform the operation as a true monobloc operation, it would be necessary to remove the costal cartilages and to reapproximate the proximal end of the ribs to the sternum. This distinction, however, is more a matter

of rhetoric than of surgery. Rarely, if ever, does a surgeon find it necessary to excise all the structures over the chin to deal properly with a cancer of the lower lip—and so similarly here; there is no need to excise ribs to meet the rigid tyranny of a definition. With the divided sternum pulled upward and laterally (the surgeon going over to the opposite side of the table for the purpose) and with the clavicle turned out of the way, ready access is had to the cervical origins of the internal mammary artery and vein and the accompanying lymphatic chain. These vessels are interrupted and the dissection is continued in a downward direction, removing as well, adjacent fat, pleura, and endothoracic fascia for 2 to 3 cm. on either side of the mammary vessels. In the intercostal spaces, the excision is extended widely beyond the borders of the lymph node chain, leaving only the superficial fibers of the external intercostal muscles intact. Moreover, this technique permits excision of the internal mammary lymph node chain throughout its entire length, including also the lymph node constantly present and not infrequently the seat of metastatic cancer at the origin of the internal mammary artery from the subclavian artery.

In reality, the operation is performed as 4 dissections with 4 resultant excisions of specimens: (1) breast and axillary contents, (2) internal mammary artery and vein accompanying lymph node chain, (3) an upper mediastinal dissection removing the portion of thymus and the fat and lymph node bearing tissue adjacent to the innominate veins, and (4) a low supraclavicular neck dissection, removing all the lymph node bearing tissue inferior to the omohyoid muscle with simultaneous clearing of the internal jugular vein and the site of confluence of jugular, subclavian, and innominate veins.

Temporary tube drains attached to a source of suction are left in the axilla and in the pleural cavity. Patients may be out of bed walking the day following operation, and the convalescent period is not much longer than following the conventional radical operation. The experience of this clinic has been that postoperative roentgen therapy offers the patient with breast cancer and lymph node metastases some increased protection. Hence we continue to advise it.

Should this extended operation be done for all cases of breast cancer? It is a good question. We do it now in all medial malignant lesions of the breast and in all patients with axillary metastases demonstrable at the time of operation. It is the current feeling of surgeons in this clinic who have performed the operation that it is a procedure which can be done with essentially the same risk as that of the radical breast operation which Halsted standardized.

What Is to Be the Surgeon's Point of View?—Only the future can tell how much additional protection this extended operation has to offer the patient with breast cancer over that provided by the conventional radical operation. Strangely enough, efforts directed at extending operations for cancer have evoked more protests from surgeons than from all other medical specialists who play important roles in the management of cancer. Some of these surgeons would have us believe that cancer surgery already had reached the zenith of the attainable prior to World War II and that surgical endeavors like that

described herein are but vain and futile gestures. A few of our surgical knights-errant, exhibiting even greater evidences of skill with a sharp pen than with the scalpel and obviously deriving keener satisfaction from use of the former and from pronouncements delivered from the lecture platform, continue to hurl volleys of verbal grapeshot and winged darts in the direction of those who believe that the surgery of cancer can be extended. Such harassing tactics should not discourage surgeons intent upon lending improvement to current accomplishments in cancer surgery. Until the promised help from other sources is near enough that evidences of such assistance can be seen or felt, I would say: let us plod on. And while we strive to extend the reach of operations, let us not forget the great importance of focusing interest and attention upon early recognition of cancer. How regrettable it is that in 1957 we cannot detect the presence of cancer in the breast until a lump is palpable. Vigilance and continued search for better devices of cancer detection are equally as important as enlargement of cancer operations. Surgeons have a dual responsibility: improvement of the techniques of recognizing as well as of treating cancer. Breast cancer is no exception.

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ABANDONMENT OF BANDING AND WIRING FOR ANEURYSM

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THE combined operation of progressive constrictive occlusion of the abdominal aorta with wiring and electrothermic coagulation was developed to meet the need for an operative technique for the management of the arteriosclerotic aneurysm of the abdominal aorta, which was beginning to become more prominent in the early 1940's. This operation was achieved in two stages, preferably one year apart. The fact that the majority of these aneurysms originated distal to the renal arteries made it feasible that the aorta could be completely occluded at a second operative stage and the blood in the aneurysm below completely clotted. Under these circumstances collateral circulation proved to be adequate

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for reasonable activity in the average patient. This feature did not entail too much handicap because activity was restricted anyway in many of the patients because of their cardiac status. Technique of procedure was particularly tedious in the first operative stage in that the adjustment of a wide slingshot rubber band wrapped in many layers of polythene containing the irritant, dicetyl phosphate, was difficult to apply to the aorta to obtain just the right degree of occlusion. The irritant action of the dicetyl phosphate served to promote sufficient fibrosis in the aorta beneath the band to prevent cutting through the wall of the aorta and causing hemorrhage. In patients in whom complete occlusion of the aorta and the aneurysm were finally obtained, the long-term results were very good. In patients in whom there was some blood continuing to flow through the aneurysm, there was a tendency for the aneurysm to grow gradually over the ensuing years. A good feature of the procedure was that it could be achieved on patients, many of whom were poor operative risks, at a reasonable mortality rate. The operative mortality on the last 32 patients operated upon was 10 per cent.[1]

With the coming of the ideal operation for aneurysm, namely, excision with restoration of arterial continuity by prosthesis, we have discontinued the use of wiring and banding. Our present choice of prosthesis for restoration of continuity of aterial flow is the prosthesis made of Orlon or Vinyon-N plastic. I do not think that there has been sufficient time or experience to determine as yet what the best prosthesis or graft really is.

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Case Reports

PERFORATION OF DUODENAL ULCER IN THE NEWBORN WITH OPERATION AND SURVIVAL

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PEPTIC ulceration of the duodenum and stomach in infancy, first recognized by Cruveillier[3] in 1829, is established as a clinical entity. Its etiology is not known and early diagnosis, before complications ensue, is difficult.

It has been suggested that the incidence of peptic ulceration in infants and children may be 0.14 per cent based on autopsy statistics.[4] Peptic ulceration in infancy, however, usually is noted only when its complications occur. The ulcer manifests itself as hemorrhage and/or perforation usually without premonitory signs.[11] In the instance of perforation early diagnosis may be confirmed by roentgenologic examination.

Surgical closure of perforated ulcer in infants with survival has been reported. Kellogg,[6] in 1951, described a successful closure of a perforated gastric ulcer in an infant in the fourth day of life; Beattie and Bohan[1] (1952) reported a perforated gastric ulcer closed at eighty-five hours after birth. Bird[2] (1941) has described a patient surviving a perforated duodenal ulcer when surgical closure was done as early as thirty-four and one-half hours after birth; perforation apparently occurred twenty-seven hours after birth.

Perforation may occur at any age; Lee and Wells[7] reported a case of gastric ulcer which perforated in utero.

Perforations of the stomach due to defects, presumably congenital, of the musculature of the stomach wall (not ulceration) with surgical closure and survival have been reported.[8]

In view of the comparative rarity of reported survivals of duodenal ulcer perforation very early in the neonatal period, the following case is presented as perhaps the youngest infant surviving duodenal ulcer perforation with surgical closure.

CASE REPORT

V. J. C. was born at the University of Kansas Medical Center on March 25, 1954. She was a full-term, Negro girl delivered by a 17-year-old primipara in an apparently normal delivery without instrumentation. There was no unusual trauma associated with delivery. Her birth weight was 2,835 Gm., her height 47 cm., and her head circumference 29.5 cm. Respirations were spontaneous and the child appeared to be normal in all respects. Examination by a pediatrician at three hours of age disclosed an apparently

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normal newborn infant. The child was fed glucose water twice during the ensuing twenty-four hours. At the age of twenty-six hours the pediatrician was called to see the baby because of vomiting and abdominal distention which began at the age of twenty-five hours. The vomitus was clear and green colored. At the time of the examination the baby had marked abdominal distention, no bowel sounds were heard, and rectal examination disclosed a small quantity of meconium. The red blood count was 5,380,000 and the hemoglobin was 18.4 Gm. per cent. The white blood count was ~~25,000~~; polymorphonuclear leukocytes 81 (filamented 70, nonfilamented 21), lymphocytes 10, monocytes 8, metamyelocytes 1. An upright roentgenogram of the abdomen and chest disclosed free air under the left diaphragm. A diagnosis of perforated viscus possibly due to perforated ulcer was made. At the age of thirty hours a laparotomy, with cyclopropane anesthesia, was done following a saphenous vein cut down for administration of blood and parenteral fluids. A transverse incision was made in the right upper abdomen. On entering the peritoneal cavity bubbling sounds could be heard and a perforation was found in the anterior portion of the proximal duodenum just distal to the pylorus. This perforation, resembling a perforation of an acute duodenal ulcer in an adult, had shaggy edges and measured 0.3 cm. in diameter. Through this perforation were passing bubbles of air and fluid which was clear and yellowish-green in color. The perforation was closed with an omental plug and 3 simple sutures of fine silk. Further exploration of the abdomen showed no other abnormalities. The wound was closed with interrupted No. 36 stainless steel sutures through peritoneum and fascia in a figure-of-eight manner. The skin was closed with interrupted No. 4-0 silk sutures. The wound was dressed with collodion. During the procedure the infant received 60 c.c. of blood. The infant appeared to withstand the operation well.

Postoperatively the patient was treated with intravenous glucose in water, intravenous Terramycin, and continuous gastric suction. Quantities of 35 to 60 c.c. of clear yellowish gastric secretions were recovered daily for the first three days, at the end of which time the gastric suction was discontinued. On the third postoperative day gastric secretions showed 0.0 degrees of free hydrochloride and 12 degrees total acid without stimulation. Oral feedings of half-strength infant formula were instituted at this time. Aqueous penicillin, 50,000 units every four hours, was discontinued on the seventh postoperative day. On the seventh postoperative day gastric secretions showed 6.0 degrees free hydrochloride and 42 degrees total acid without stimulation. The patient's postoperative course was uncomplicated and she was afebrile except for one occasion on the eighth day when the temperature rose temporarily to 101.8° F.

A roentgenologic examination of the abdomen on the fifth postoperative day showed no free intraperitoneal air. On the tenth postoperative day a fluoroscopic examination of the chest and abdomen did not show any abnormality. On the sixteenth postoperative day a barium examination of the upper gastrointestinal tract showed a duodenal bulb which was small and much deformed. The barium passed normally through the small bulb. There was no evidence of obstruction.

At the time of her discharge from the hospital on the seventeenth postoperative day the patient was eating well and gaining well. Her weight at that time was 3,030 Gm. This represented a gain of 195 Gm. At this time the hemoglobin was 11.6 Gm. per cent and the white blood count was 17,700.

Three weeks postoperatively the patient was readmitted to the hospital because of a history of pink-tinged stools of two-days' duration. Examination showed a normal-appearing child. A guaiac test of the stools for blood was reported as 4-plus. Roentgenologic examination of the abdomen was normal. The hemoglobin was 11.2 Gm. per cent. The gastric secretions showed 2.0 degrees of free acid and 8.0 degrees of total acid. The stools became negative for blood after two days.

At the age of six months the patient was admitted to the hospital for repair of a large umbilical hernia. This operation and postoperative course were uneventful. The hemoglobin at this time was 10.8 Gm. per cent. An upper gastrointestinal roentgenologic series showed slight deformity of the duodenal bulb but the stomach emptied well. The child weighed 6,790 Gm., which represented a gain of 3,950 Gm. from its birth weight.

The child was last seen in the surgery clinic at the age of sixteen months. She was in excellent health and on examination she was considered to be normal in every respect.

COMMENT

Although preoperative diagnosis was only suspected in this case, it is believed that the most important factor in the management of this infant was the early attention to the patient's first signs of abdominal distention, which led to an early upright roentgenologic examination. The obvious radiologic evidence of perforation resulted in early operation, only thirty hours after normal birth, and four or five hours after suspected perforation.

It will be noted that vomiting of material not containing blood occurred early in the history of this infant; however, it was the distention, more than the vomiting, which alerted the pediatric resident to prompt recognition of the serious nature of the condition and surgical consultation.

In the usual absence of premonitory symptoms or signs, early diagnosis of perforated ulcer depends upon close observation of newborn infants for abdominal distention, refusal of oral intake, and vomiting. Hematemesis or melena, beginning deterioration, and marasmus follow the earlier signs of this catastrophe.

The rather marked changes in the gastric secretion of acid within the first twenty-four hours of the neonatal period of infants are interesting. Immediately after birth the gastric contents are neutral in reaction but within a few hours there is an excess of acid with measurements of pH which indicate a strongly acid reaction between 3 and 1 (average of 1.45) sufficient to activate pepsin, which usually is present at this time.[5] This acidity is equivalent to 36 ml. N/10 HCl which agrees with the extensive studies of Miller,[9] who found in a large series of infants that the average acidity was about 27 ml. of free acid. He also stated that the degree of the acidity seems to be roughly related to the gross quantity of gastric juice secreted; usually more juice can be withdrawn from those infants in whom the reaction is most acid. The average volume aspirated in normal newborn infants is stated to be 4.5 ml. After the prompt postnatal increase the peak of gastric acidity tends to decline until about the eighth day, with some increase in the second week of life.[10]

In the infant presented here, rather large quantities of gastric secretions were aspirated postoperatively (35 to 60 c.c. net daily for the first three days), although qualitative measurements of acidity on the third, seventh, and twenty-second postoperative days were not considered abnormal.

It is of interest to note that this child bled from the gastrointestinal tract for a period of three to four days, three weeks after surgical closure of the ulcer. Cessation of bleeding was spontaneous and the child has continued to gain normally.

SUMMARY

A case is presented which calls attention to early recognition of perforation, a complication of duodenal ulcer in the neonatal period. In this infant, perforation occurred about twenty-five hours after a normal birth, and surgical closure was done at the age of thirty hours, with survival of the patient.

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INTESTINAL OBSTRUCTION DUE TO ENTEROLITH COMPLICATING JEJUNAL DIVERTICULOSIS

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MULTIPLE diverticula of the jejunum constitute a developmental abnormality, occurring in about 0.003 per cent of otherwise normal individuals. The condition is generally symptomless, and is found in the course of a laparotomy for some unconnected condition, or in the course of autopsy. That it can give rise to symptoms as a result of complications has, however, long been recognized, and these were brilliantly reviewed by Professor Milnes Walker[6] in 1945.

The commonest complication is acute infection of one or more diverticula, with the natural sequence of perforation, abscess formation, and peritonitis. Occasionally the accumulation of fecal material in one of these diverticula is extruded into the lumen of the bowel in the form of an enterolith, and should this be of large caliber, acute intestinal obstruction may supervene. How often such enteroliths are extruded and normally passed in the stools is of course impossible to say, but that such is of frequent occurrence is suggested by the fact that intermittent attacks of colicky abdominal pain have been recorded so often in patients suffering from these diverticula that they are included in its symptomatology.

The present case is of interest in that, in addition to acute obstruction having been caused by the impaction of one such enterolith, the diverticula were not confined to the jejunum, but were present throughout the alimentary tract from the duodenum to the sigmoid colon. Radiology demonstrated a large one in the duodenum, and the barium-filled colon gave the identical picture that one finds in very severe colonic diverticulitis, a condition which is not usually considered to be of congenital origin.

CASE REPORT

M. H., aged 56 years, was admitted to Upton Hospital on Feb. 26, 1955, as an abdominal emergency. Her health had always been good until seven weeks before admission when she had had an attack of acute colicky abdominal pain, followed by vomiting. She was diagnosed as a case of "gastric flu," and treated with rest in bed. The symptoms soon subsided, but these attacks recurred at intervals throughout the next seven weeks, until her present attack occurred, which was similar to her previous attacks, but very much more severe.

On admission her abdomen was distended and visible peristalsis was present. A diagnosis of acute, supervening on chronic, small bowel obstruction was made, and she was taken to the theater. On opening the abdomen, the first thing noticed was the enormous number of jejunal diverticula present, varying from the size of a thimble to that of a golf ball. The obstruction was low in the ileum, and was due to an impacted foreign body. This was removed, and consisted of a flattened, oblong, yellowish stone, measuring 2½ by 1½ inches

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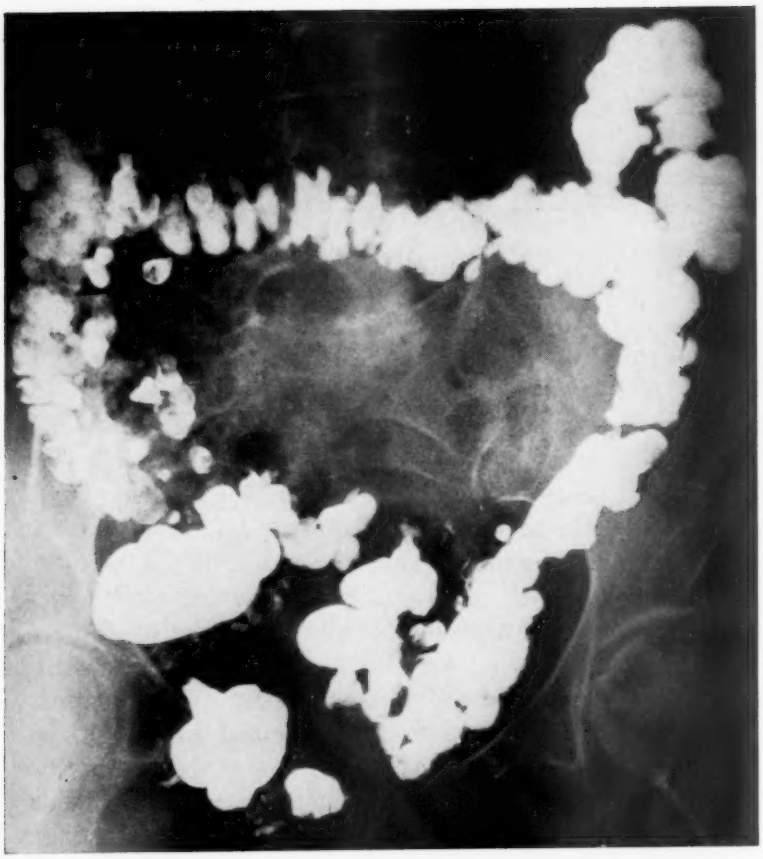

Fig. 1.—Postoperative x-ray film showing multiple colonic diverticula.

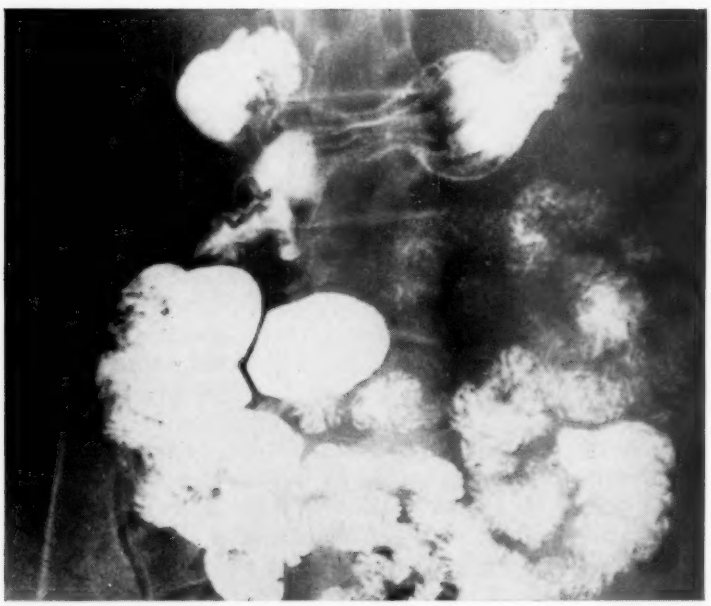

Fig. 2.—Postoperative x-ray film showing large duodenal diverticulum and multiple large jejunal diverticula. The latter are completely opaque while the normal jejunal lumen is only partially so.

by 1 inch. Naturally, one's first impression was that this was a case of gallstone ileus, but gallbladder and bile ducts were perfectly normal, and it was difficult to believe that a stone of this size could have developed from a normal biliary tract.

Postoperative progress was uneventful, and the patient was discharged on March 12. Examination of the stone showed it to consist of calcium and phosphate, with a small quantity of cholesterol, and to be a true enterolith. A subsequent barium series revealed an amazing sequence of diverticula involving the whole of the small and large intestine from duodenum to sigmoid colon.

I should like to thank Drs. Emmerson and Shalom for their assistance with this case, and Mr. Raymond Ramsay for permission to publish.

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CONGENITAL ABSENCE OF THE GALL BLADDER

Report of a Case

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(From the Sanders Clinic)

THE findings encountered in a recent surgical procedure stimulated our interest in the relatively rare developmental anomaly of congenital absence of the gall bladder. In discussing congenital absence of the gall bladder, we refer only to those cases in which the absent gall bladder is the only defect of the extrahepatic biliary system, and exclude those cases in which there is any associated extrahepatic biliary atresia. We have reviewed the literature on this subject, and found 104 cases which have been reported in the literature since 1900.

Gross,[11] in 1936, reported 38 cases of this anomaly which had appeared in the literature since 1900. In 1945, Dixon and Lichtman[8] analyzed 50 cases, and added 10 cases from the records of the Mayo Clinic. These authors mentioned 30 additional cases reported prior to 1900, as well as a number reported since 1900, in which the clinical information was too scant to justify inclusion in their analysis. In 1947, the total number of cases in the literature had risen to 71, as reported by Latimer, Mendez, and Hage,[15] who presented three additional cases. Since their article, 30 cases have been reported,[1-7, 9-14, 16-31] a number of which are in foreign journals. The information regarding some of these cases is incomplete, yet a definite case presentation was made in each.

CASE REPORT

Mrs. F. C., a 32-year-old white woman, was first seen on Sept. 17, 1953, requesting a general examination. She had numerous complaints, one of which was indigestion of several years' duration. This occurred daily, and was manifested by cramping, upper abdominal pain, a sensation of bloating in the abdomen, and excessive belching. These symptoms were aggravated by eating greasy, fried, or highly seasoned foods. She had had some constipation for many years, but no change in bowel habits.

The system review was negative except for a sensation of tightness in the throat and some occasional headaches.

Physical examination revealed a well-developed, well-nourished woman. The blood pressure was 104 mm. Hg systolic, with a diastolic pressure of 60 mm. Hg. Temperature and pulse were normal. Physical examination was entirely negative otherwise.

Except for a slight anemia and a few white cells in the urine, the routine laboratory procedures were normal.

Chest roentgenogram was negative. The stomach roentgenogram showed slight spasticity of the duodenal cap, and the cholecystogram was reported as showing nonfunction of the gall bladder.

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She was placed on a liberal ulcer diet, given antispasmodic drugs, and told to return in two months for revaluation and repeat roentgenogram of the gall bladder.

She returned on Nov. 20, 1953, at which time a repeat cholecystogram again showed nonfunction. In view of this finding, and the lack of improvement on antispasmodics, cholecystectomy was advised.

At surgery, the gall bladder was entirely absent, and in the area which the gall bladder normally occupies there was a 2 mm. wide, white, fibrous thickening of Glisson's capsule. Superficial dissection was done in this area to see if there was any evidence of an intrahepatic gall bladder but none was found. The common duct was not opened. The liver was entirely normal. Following thorough abdominal examination and appendectomy, the wound was closed.

The patient made an uneventful recovery, but for several months she continued to have the same complaints which she had had preoperatively.

X-ray study of the biliary tract on July 28, 1954, using Cholegrafin (Fig. 1) revealed the common duct to be normal, with no evidence of a gall bladder or remnants thereof.

At the present time her gastrointestinal complaints have improved, but have not entirely cleared.

Fig. 1.—Cholegrafin study showing common bile duct well outlined. No evidence apparent of ectopic gall bladder or other anomalies.

DISCUSSION

Congenital absence of the gall bladder is a relatively rare anomaly. The presence of the symptoms usually attributed to cholecystic disease is not necessarily due to a diseased gall bladder, as 18 of the 30 cases in the literature since 1947 did have symptoms compatible with the usual diagnosis of "chronic

cholecystitis with stones." Two others had symptoms not typical but still suggestive of cholecystic disease. In the remaining 10 cases, the history was not sufficiently detailed to determine the symptomatology.

A preoperative diagnosis of the anomaly is impossible, as there is no known procedure to differentiate absence from nonfunction of the gall bladder. A preoperative cholecystogram was reported in 16 of the 30 cases since 1947. Fourteen of these were interpreted as nonfunction of the gall bladder with no stones visualized. Two cases were interpreted as "faint visualization" of the gall bladder.

In recent years, the great majority of cases have been found during a surgical procedure for suspected cholecystic disease. When this condition is encountered at surgery the surgeon must make a thorough exploration in an attempt to eliminate the possibility of anomalous location of the organ. The presence of an intrahepatic gall bladder is difficult to completely exclude. An operative or postoperative cholangiogram is probably the best method of achieving this, but it seems questionable to us whether the retrograde dye would flow into a nonfunctioning intrahepatic gall bladder, particularly if a stone were impacted in the cystic duct.

An interesting observation is that made by Kobacker[13] in his presentation of two sisters with congenital absence of the gall bladder, in which he suggested the possibility of an hereditary defect. He went a step further and made x-ray studies of 5 siblings of these two sisters; three of these showed nonfunction of the gall bladder. This suggested they too might have this anomaly.

SUMMARY

1. A case of congenital absence of the gall bladder is presented.

2. A survey of the available literature revealed 104 cases of this anomaly reported in the literature since 1900.

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Erratum

Unfortunately an error was made in the editorial entitled "Is the Treatment of Benign Thyroid Disease With Radioactive Iodine Without Danger?" (December, 1956, issue of the JOURNAL, page 1129) in that in quoting the study of Simpson, Hempleman, and Fuller percentages were used rather than the number of cases. The fact that the figures show a comparison between the expected incidence and the observed rate demonstrates the possible danger of irradiation, although the total incidence is a good deal less when "percentages" are deleted than was stated in the editorial. The statement should have read: "The expected incidence in all cases in both the irradiated and the nonirradiated groups was 2.6 whereas the observed incidence in the former was 17 and possibly 19 and in the latter was 5. The expected incidence of leukemia in both groups was 0.6, whereas the observed incidence was 7 and possibly 8 in the irradiated and none in the nonirradiated group. The expected incidence of thyroid cancer in both groups was 0.08, whereas the observed incidence was 6 in the irradiated group and none in the nonirradiated group."

Book Reviews

CONDUCTED BY J. GARROTT ALLEN, M.D.

The Abnormal Pneumoencephalogram. By Leo Davidoff and Bernard S. Epstein. Ed. 2. Philadelphia, 1955, Lea & Febiger. Pp. 518.

The first edition of this book was published in 1950 and only minor changes have been made in this second edition. For the most part these changes are the citations of additional literature, none later than 1953 and most refer to literature published between 1949 and 1953, inclusive.

In addition a few new sentences have been added and others revised. Two additional figures are included (Fig. 142, page 271 and Fig. 291, page 486). One new case report has been added (Case 116).

The authors' material is now stated to cover a 24-year period instead of the 20-year period covered in the first edition, but 500 cases segregated for statistical analysis are obviously the same cases analyzed in the first edition.

Correct reference is now made to Fig. 182*A* to illustrate the fingerlike collection of gas in the diagnosis of subdural hematoma, whereas in the first edition reference was erroneously made to another figure.

Laminagraphy as part of the pneumoencephalographic examination to demonstrate certain lesions, especially those that are parasellar or in the midline, is advocated repeatedly in both editions. As the value of this procedure is now becoming more widely appreciated it is more extensively used.

—William T. Peyton

Pelvimetry. By Herbert Thoms. New York, 1956, Paul B. Hoeber, Inc. 8° cloth, 120 pp., 45 ills., 3 tables. $5.00.

Accurate pelvic evaluation is essential for the intelligent conduct supervision of patients in labor. Many mothers and infants are subjected to unnecessary hazards because of the obstetrician's failure to recognize or correctly to interpret the significance of variations in pelvic morphology and capacity.

This authoritative, concise, well-illustrated monograph deals with the practical aspects of pelvic evaluation. The author is of the opinion that every primigravida should have an accurate pelvic survey employing routine x-ray pelvimetry in addition to the customary clinical evaluation of pelvic adequacy. He feels that the position of those individuals who contend that suitable screening can be accomplished by clinical examination alone is unsound since important inlet and midpelvic dimensions cannot be accurately evaluated by internal clinical pelvimetry.

A single inlet film is advocated for screening purposes. This reveals the morphology of the inlet, midplane, and outlet. In addition, accurate measurements of the transverse diameters of the inlet, the midplane, and the anterior-posterior diameters of the inlet and outlet can be made from this film. More detailed roentgenologic study is indicated if unusual pelvic morphology is seen or is suspected.

Normal and abnormal pelvic variations and the modern concepts of midpelvic morphology are described and their significance in the conduct of labor is discussed. The chapters dealing with pelvic development, the influence of nutrition on pelvic conformity, injuries to the coccyx, separation of the symphysis, and the technique of x-ray pelvimetry add to the value of this timely monograph.

—Charles P. McCartney

Anesthetic Accidents. By V. Keating. Chicago, 1956, The Yearbook Publishers, Inc. Pp. 253. $5.00.

This fine little book is, at first glance, a little frightening. Those who give anesthesia regularly rarely stop to enumerate all of the things that might happen to the patient under his care, and to see them all listed together takes one aback.

Dr. Keating has collected from his own and the experience of others a large number of accidents and complications which occur during or as a result of anesthesia. These have been grouped under such general headings as "Complications of Local Anesthesia," "The Effects of Position in Anesthesia," and "The Side Actions of Relaxants." Two chapters relate purely to the effects of general anesthesia on circulation and respiratory function. Interestingly, the author has given much space to the physiologic and pharmacologic background for anesthetic complications. He states in his foreword that he has given more prominence to the work of the physiologist and pharmacologist than he had first intended. Problems relating to the mechanical aspects, such as explosion, broken needles, and equipment failure, are also described.

Besides giving a good description of various anesthetic accidents and their causes, the author discusses well what to do when the unforeseen happens. The bibliography, though probably not complete, is extensive and the discussions are well documented. The book is a handy size, well bound, and printed in a very legible type face. All in all, it's a book that ought to be thoughtfully read and kept within easy reach by the practicing anesthetist, the sometime anesthetist, and the surgeon.

—Donald Benson

Clinical Orthopaedics. Anthony DePalma, M.D., Editor in Chief. Vol. VIII. Philadelphia, 1956, J. B. Lippincott Company.

The *Clinical Orthopaedics* series is undoubtedly familiar to the majority of orthopedic surgeons. This is the eighth such volume. It is divided into three sections which are entitled "Chronic Hereditary Diseases and Developmental Anomalies," "General Orthopedics," and "Motorist Injuries and Motorist Safety." Each section is made up of original articles by specialists in their respective fields.

The emphasis in the first section is on application of genetics to orthopedic problems. Of particular interest are articles by Stechner ("Genetics of Joint Diseases"), Johnston ("Inheritance of Hand and Foot Anomalies"), and Herndon ("Osteogenesis Imperfecta: Some Clinical and Genetic Considerations"). More conventional consideration of several other congenital problems is also included. Among these is a résumé by Denis Browne of his methods of management of several congenital problems in a paper entitled "Splinting for Controlled Movements."

The section "General Orthopaedics" is headlined by DePalma and Cotler's extensive paper "Hemophilic Arthropathy." This article is based on experience with 117 hemophiliac families followed at the Jefferson Medical College Hospital in a hemarthropathy clinic established under the combined direction of the Departments of Hematology and Orthopedic Surgery. The clinical course of these patients is covered as well as the x-ray changes which are observed to occur. Observations on gross and microscopic pathology of the joint disease were considerably aided by an unreported autopsy of a 27-year-old hemophiliac. A review of the classification of hemophilic arthritis is made and a new classification is presented.

Dr. Jacob Kulowski is guest editor of the third section of the book in which motorist injuries and motorist safety are discussed. This fresh subject is presented from the point of view of research which has been undertaken by the police, the engineer, the glass manufacturer, and others. Equipment and methods used in the study of auto collisions are described by Severy and Mathewson in a detailed paper, "Automobile-Barrier Impacts."

This book is primarily written for those interested in the specialty of orthopedics. Individual articles, to be sure, are of broader interest. The strength and weakness of the book rests on a common ground, the fact that it covers a multitude of subjects from as many points of view.

—Wayne H. Akeson

Diagnosis and Treatment of Peripheral Vascular Disorders. By David I. Abramson. New York, 1956, Paul B. Hoeber, Inc.

A comprehensive review of the diagnostic symptoms and signs (Part I), and the disease entities (Part II) of peripheral vascular diseases is presented almost totally from a clinical and admittedly from a nonsurgical point of view. A superficial description of anatomy, physiology, and pharmacology is given separately (Part III). The material is available for perusal as well as seeking specifics and is a good text for clinical use. "Avoiding theory and academic discussion, the author has provided here what the clinician needs to know about peripheral vascular disorders." (Publisher's leaf.) The surgeon can scan this book usefully, because there are a number of medical treatments and diagnostic aids useful to him presented from a large clinical experience. Doctor Abramson acknowledges a medical prominence and bias in the book, but his over-all experience reveals a very real acquaintance with an excellent standard of surgical practice in these disorders, and the book is only nonsurgical in that this aspect is given merely outline representation.

Many theoretical disadvantages of sympathectomy are discussed in a general way in the last part of the book in the chapter "Sympathetic Nervous System" as well as in the chapters on specific organic vascular disorders, but the author often ends up recommending the procedure as an adjunctive or for "protecting a remaining limb" in a disease where it had been labeled as not useful.

Specific items such as foot care, dosage of pharmacologic agents, and the discussion of the clinical course of frostbite are well done.

—Peter V. Moulder

Manual of Anesthesiology for Residents and Medical Students. By H. Schwartz, S. H. Ngai, and E. M. Papper. No. 298 in the American Lecture Series, Springfield, Ill., 1957, Charles C Thomas, Publisher. Pp. 170, 12 ills. $4.25.

This book is written as a guide for the beginner to be used concomitantly with his first technical training in anesthesia. As his training advances this manual becomes less important than the reading of original source material.

Through 23 short chapters the enormous subject of anesthesia is covered in an admirable, clear, and brief way without being reduced to a truncate enumeration. Selected references will guide the student, after a few weeks in anesthesia, into more detailed and specific studies.

Respiration and circulation occupy rightfully a major portion of the book, and clinically important factors are stressed with regard to physiology and understanding, as to clinical anesthesia and resuscitation.

The presentation is brief, clear, and easily read, and an extensive index facilitates the usefulness during the first clinical training of students, interns, and residents. The 12 illustrations are schematic and easily understood; more illustrations would increase the value of the book for the beginner.

The book is printed on a good quality of paper in a pleasant type, and is well bound.

The authors have mastered the difficult task of writing a brief, easily read book covering a vast field, and undoubtedly it will be of great help to the medical student, the intern, and resident during his first and often fumbling experiences in anesthesiology.

For the instructor, this manual is useful in the organization of the training of the beginner.

—E. Trier Mörch

The Cellular Basis of Wound Repair. By Martin Allgower. Springfield, Ill., 1956, Charles C Thomas, Publisher.

Allgower's monograph covering a long set of experiments on wound healing reads with the tension and fluidity of a novel. The unfolding of a series of crucial experiments with sufficient detail and reference to allow repetition and enough data to represent fact finds the nuclear blood cell as the unequivocal source for the granulation tissue of wound repair. The book is humbly presented as re-establishing the findings of Alexander Maximow in the early part of the century, but with so few aware of these facts and the implications of the true totipotency of leukocytes and their progenitors as established by the school of Maximow and Bloom, this is a refreshing reawakening. Granting this priority does not detract from the strength of this work as it is done from a different viewpoint (wound healing) and with many new techniques. Data to establish the fact of the blood cell source are presented from observations on surgical wounds, per se, with colchicine mitotic blockade, wounds in leukopenic (irradiation-produced) animals, and tissue and cell culture media with in vivo and in vitro (histologic) data. Isolated, perfused rabbit ear experiments failed to be useful because of the rapid appearance of edema. Observations on the quantitative aspects of wound repair with the addition of "known" leukotactic substances showed no variation from the controls.

These are excellent, exciting experiments presented with good photomicrographs that can be readily deciphered. One hesitates to note minor flaws, but as such they emphasize the solidity of the main thesis. Reference to control data on wounds and culture media without added penicillin and other materials is not given; the irradiation experiments could be pushed further with lethal one-dose total body irradiation; and the author seems to accept the Maximow cell source without recognizing the broad concepts of this theory.

—Peter V. Moulder

Lesions of the Cervical Intervertebral Disc. By Glen Spurling. Springfield, Ill., 1956. Charles C Thomas, Publisher, xi, 134 pp., 41 ills. $4.75.

Pain in the neck, with or without radiation into the arm, exists in a field of confused ideas and multiple treatments. It is not many years since the scalenus anterior muscle was considered of major etiologic importance. At the present time most medical experts would recast in that role the cervical intervertebral disc, and with these I would concur. This short book gives an account of the anatomy and possible pathology of the cervical disc together with a history of the development of present concepts. More important, it presents the therapeutic conclusions of a surgeon of sound judgment and extensive experience in the field.

The problem presents in 3 clinical groups: (1) the patient with much neck pain and minor radiation in the arm, (2) the patient with unequivocal single nerve root compression, and (3) the patient with long tract signs and little trouble referable to the neck save the presence of radiographic osteophytes and myelographic bars. With the first group the author unfortunately deals but little. They are the people not sufficiently ill to require traction in a hospital but feel too much pain to work in comfort. His conservative and surgical management of the second group is generally accepted and widely employed but the surgical management of the third is not yet fully accepted either in theory or in practice. The role played by the dentate ligaments is debatable, particularly when we consider the gross distortion of the cord and of the ligaments that may occur in association with tumor, angulating kyphosis, and collapse of vertebrae without comparable clinical signs.

This book contains much valuable advice on operative technique such as the admonition to refrain from vigorous surgical excavations anterior to the cord. Though the problems of the cervical disc are not yet as clearly understood as those of the lumbar disc, this book is a significant contribution to their elucidation and deserves a place beside the author's well-known monograph, *Lesions of the Lumbar Intervertebral Disc.*

—John F. Mullan

Training of the Lower Extremity Amputee. By Donald Kerr and Signe Brunnstrom. Springfield, Ill., 1956, Charles C Thomas, Publisher.

This book presents a concise treatment of the problems created by amputation from the postoperative period onward to their culmination in the ultimate inspiring achievements of many amputees in occupational and recreational activities. In this manual will be found sound coverage of the preprosthetic period as well as detailed description of training methods designed so that maximum efficiency in the use of the limb will be achieved by the amputee. A brief description of various types of lower extremity prostheses is included. Considerable detailed information is offered which is of value to the physical therapist working with amputees as well as to the amputee himself. The book is also of value in a more general sense to the resident or specialist who manages amputees, presenting an excellent review of the scope of the problems which arise once surgery is completed.

Full effort has been made by the authors to include all the combinations of possibilities which may occur as regards the level of amputation. There are chapters dealing with the single above-knee amputee, the "tilting table" amputee, the single and double below-knee amputee, the double (one above- and one below-knee) amputee, and the double above-knee amputee. In each case the chapter is concluded by a section on practical hints for daily use in which the myriad of details of daily life which are altered by the amputation are considered.

The book is based on the experience of an amputee, Mr. Kerr, now Director of the National Institute for Amputee Rehabilitation, and Miss Signe Brunnstrom, a physical therapist now in charge of amputee training at the Institute of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center.

The book is well organized, quite readable, and superbly illustrated. It is the product of that rare gifted author who has command of his subject matter and who can at the same time present the material to others. It is a welcomed, well worth-while addition to its field as well as a stand out example of a piece of good writing.

—Wayne H. Akeson

ANNOUNCEMENT

The Foundation of the American Society of Plastic and Reconstructive Surgery, Inc.

1957 SCHOLARSHIP CONTEST

I. *Junior Classification.*—

1. Restricted to residents in training and plastic surgeons in practice no longer than 5 years; must be residents of American continents, British Dominions, or Western Europe.

2. *First award:* 6 months' scholarship; *second award:* 3 months' scholarship. Award winners will be entitled to full maintenance in all plastic surgery services listed in the Foundation's pool in the United States and Canada. Limited maintenance will be available in all foreign plastic surgery centers officially listed in the pool. Scholarship time may be divided to suit the requirements of the winners. Additional cash prizes of $1,000.00 and $750.00 will be made available to the first and second award holders, respectively, for traveling expenses.

3. Certificates of Honorable Mention may also be given, with or without a limited scholarship.

4. Character references by two leading plastic surgeons from the applicant's country must accompany the manuscript.

II. *Senior Classification.*—

1. Contestants must be in active practice of plastic and reconstructive surgery for more than 5 years.

2. Award: Silver Plaque or Certificate of Honorable Mention for winning essay.

III. *Original Contributions in Plastic Surgery Field Submitted by Nonplastic Surgeons.*—

1. Award: Silver Plaque or Certificate of Honorable Mention for winning essay.

Rules Applicable to All Contestants

Manuscripts should be sent by registered mail and will not be accepted by the Award Committee after June 1, 1957.

The subject matter of the essay must be the result of some original research (either clinical or experimental) in plastic and reconstructive surgery and about 5,000 words in length. Papers previously published (even in part) will not be accepted.

All essays shall be submitted in quadruplicate, in English only, *with no indication of the writer's name or institutional affiliation.* An additional envelope shall include contestant's name, address, and institutional affiliation, with details of professional training and percentage of practice in plastic surgery.

Award winners shall present a written report before their national plastic surgery societies on their scholarship experiences. The report should be published in their plastic surgical journals.

The Foundation of the American Society of Plastic and Reconstructive Surgery, Inc., reserves the right to publish the submitted essays in the official Journal, *Plastic and Reconstructive Surgery.*

Infraction of any of the above listed regulations will prevent participation in the contest.

Send all manuscripts to Dr. Richard B. Stark, 20 East 68th St., New York 21, N. Y.

For further information write The Foundation of the American Society of Plastic and Reconstructive Surgery, Inc., c/o Dr. C. R. Straatsma, President, 5 East 83rd Street, New York 28, N. Y.

Calendar of Surgical Meetings

1957

AMERICAN ACADEMY OF NEUROLOGICAL SURGERY. *Annual Meeting,* Nov. 11-13, 1957, The Cloister, Sea Island, Ga. Deadline for papers, Aug. 15, 1957. Secretary, Dr. Eben Alexander, Jr., Bowman Gray School of Medicine, Winston-Salem, N. C. Attendance at meetings by invitation only.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY. *Annual Meeting,* May 4, 6, and 7, 1957, The Palmer House, Chicago, Ill. Deadline for papers, Dec. 15, 1956. Secretary, H. T. Langston, M.D., 1919 West Taylor St., Chicago 12, Ill.

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS. *Annual Meeting,* May 1, 2, and 3, 1957, The Homestead, Hot Springs, Va. Secretary, John A. Taylor, M.D., 2 East 54th Street, New York 22, N. Y.

AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA. *Annual Meeting,* Oct. 31, Nov. 1 and 2, 1957, The Homestead, Hot Springs, Va. Deadline for papers, Aug. 1, 1957. Secretary, James K. Stack, 700 N. Michigan Ave., Chicago 11, Ill.

AMERICAN CANCER SOCIETY. *Annual Meeting,* Oct. 28-29, 1957, Park-Sheraton Hotel, New York City, N. Y. Participation by invitation only.

AMERICAN COLLEGE OF SURGEONS. *Clinical Congress,* Oct. 14-18, Convention Hall, Atlantic City, N. J. Secretary of the College, Michael L. Mason, 154 E. Erie St., Chicago 11, Ill.

THE AMERICAN GOITER ASSOCIATION. *Annual Meeting,* May 28-30, 1957, Hotel Statler, New York, N. Y. Secretary, John C. McClintock, M.D., 149½ Washington Ave., Albany 10, N. Y.

AMERICAN GYNECOLOGICAL SOCIETY. *Annual Meeting,* May 27, 28, and 29, 1957, The Homestead, Hot Springs, Va. Secretary, Andrew A. Marchetti, M.D., Georgetown University Hospital, Washington 7, D. C.

AMERICAN MEDICAL ASSOCIATION. *Annual Meeting,* June 3-7, 1957, Coliseum, New York, N. Y. Deadline for papers, Dec. 15, 1956. *Clinical Meeting,* Dec. 3-6, 1957, Auditorium, Philadelphia, Pa. Deadline for papers, July 15, 1957. Secretary, American Medical Association, George F. Lull, M.D.; Secretary, Council on Scientific Assembly, Thomas G. Hull, M.D.; address of both, 535 North Dearborn Street, Chicago 10, Ill.

AMERICAN SURGICAL ASSOCIATION. *Annual Meeting,* May 8, 9, and 10, 1957, The Palmer House, Chicago, Ill. Secretary, R. K. Gilchrist, M.D., 59 East Madison Street, Chicago 3, Ill.

AMERICAN UROLOGICAL ASSOCIATION. *Annual Meeting,* May 6-9, 1957, William Penn Hotel, Pittsburgh, Pa. Deadline for papers, Jan. 1, 1957. Secretary, Samuel L. Raines, M.D., 188 S. Bellevue Blvd., Memphis, Tenn.

INTERNATIONAL SOCIETY OF SURGERY. *Seventeenth Congress,* Oct. 20-27, 1957, City University Medical School, Mexico City. Details of hotel accommodations and reservations are being handled by "Mexamericana," Paseo de la Reforma 44, Mexico, I. D. F.

NEW ENGLAND SURGICAL SOCIETY. *Annual Meeting,* Oct. 4 and 5, 1957, Mount Washington Hotel, Burton Woods, N. H. Deadline for papers, June 1, 1957. Secretary, Richard Warren, M.D., Peter Bent Brigham Hospital, 721 Huntington Avenue, Boston 15, Mass.

PAN-PACIFIC SURGICAL ASSOCIATION. *Seventh Congress,* Nov. 14-22, 1957. Honolulu, Hawaii. Director General, F. J. Pinkerton, M.D., Room 230, Young Building, Honolulu, Hawaii.

THE SOCIETY FOR VASCULAR SURGERY. *Annual Meeting,* June 2, 1957, Hotel Biltmore, New York, N. Y. Secretary, Henry Swan, M.D., 4200 E. 9th Avenue, Denver, Colo.

SOUTHERN SURGICAL ASSOCIATION. *Annual Meeting,* Dec. 10-12, 1957, Greenbrier Hotel, White Sulphur Springs, W. Va. Secretary, George G. Finney, M.D., 2947 St. Paul St., Baltimore 18, Md. Guests are invited by the Secretary at the request of the Membership.

WESTERN SURGICAL ASSOCIATION. *Annual Meeting,* November 21-23, 1957, Hotel Utah, Salt Lake City, Utah. Deadline for submission of papers, Aug. 15, 1957. Secretary, John T. Reynolds, M.D., 612 N. Michigan Ave., Chicago 11, Ill.

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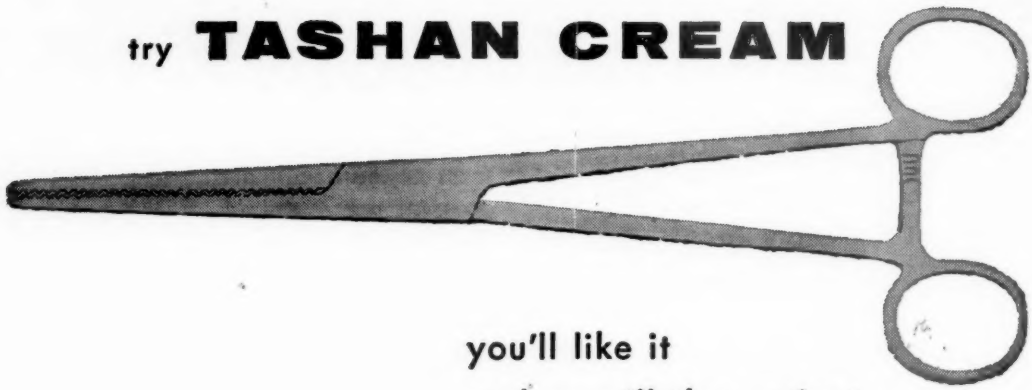

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